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13. ABSTRACT (Maximum 200 words)

The study entitled "Development of a Standard for the Health Hazard Assessment of Mechanical Shock and Repeated Impact in Army Vehicles" was designed and conducted in 5 phases between July 1991 and July 1997. The primary objective was to develop a dose-effect model to predict, and ultimately minimize, the risk of injury to a soldier when exposed to the repeated shock environment of tactical ground vehicles (TGVs). Phase 1 reviewed over 1,200 relevant scientific, medical and military papers. Phase 2 analyzed and characterized the vibration and shock environment of Army TGVs. Based on Phase 2, motion simulations were developed for the experimental phases. Phase 3, a pilot study, determined the most sensitive human response measures to mechanical shock and repeated impact. Phase 4 identified important factors (biomechanical, physiological, biochemical and subjective responses to motion exposure) to include in the development of a health hazard assessment model. In Phase 5, a health hazard assessment method was developed for mechanical shock and repeated impact in Army vehicles. A series of models were developed and programmed into a graphical user interface to simplify the application of the health hazard assessment models to measured seat accelerations. Together these models predict the risk of injury based on fatigue failure theory.

14. SUBJECT TERMS
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Executive summary

This report summarizes work conducted in Contract No.
DAMD17-91-C-1115 entitled "Development of a Standard for the
Health Hazard Assessment of Mechanical Shock and Repeated Impact
in Army Vehicles". Research was conducted by a project team
located at B.C. Research Inc. in Vancouver, B.C., Canada between
July 1991 and July 1997. Due to the length and scope of the
project, the methodology was developed in five phases throughout
the study, based on acquired knowledge and data.

The study was designed and conducted at facilities at B.C.
Research and at the United States Army Aeromedical Research
Laboratory (USAARL) in Fort Rucker, Alabama. Phase 1 reviewed
over 1,200 relevant scientific, medical and military papers. The
literature review, which supported the primary objective to
develop a dose-effect model to predict and ultimately minimize
the risk of injury to a soldier when exposed to the repeated
shock environment of tactical ground vehicles (TGVs), fills an
important gap in the scientific literature. Phase 2 analyzed and
characterized the vibration and shock environment of Army TGVs.
Mathematical and computational methods were developed to simulate
the motion of TGVs. These motion signatures were used to drive
the multi-axis ride simulator (MARS) in Phase 3 and Phase 4.
Phase 3, a pilot study, determined the most sensitive human
response measures to mechanical shock and repeated impact for use
in the experimental phase of the study. Phase 4, the
experimental phase, identified important factors (based on the
biomechanical, physiological, biochemical and subjective
responses to motion exposure) to include in the development of a
health hazard assessment (HHA) method.

In Phase 5, a method was developed for HHA of mechanical
shock and repeated impact in Army vehicles. This phase included
the development and integration of the following: dynamic
response models for mechanical shocks in the x, y, and z axes;
a biomechanical model to estimate internal forces at the L4/L5
joint; a dose model to represent cumulative stress; and a
probability of injury risk model based on population variance in
the strength of the spine. A vehicle test matrix was developed
which included a hazard severity classification, hazard
probability classification and determination of risk assessment
codes (RAC). A software version of the HHA method with a
graphical user interface (GUI) was developed to simplify the

application of the health hazard assessment models to measured seat accelerations.

Table of contents

List of illustrations

X

List of tables

Introduction

Background

The Army relies on standard guidelines to assess the effects of repeated shock and vibration on performance, fatigue and health and safety of the soldier while he operates or is transported by tactical ground vehicles (TGVs). At the outset of this project, the main standards and guidelines for human exposure to repeated shock and vibration included the following:

- International Organization for Standardization (ISO) 2631-(1978, 1982, 1985, and 1989)

- British Standard (BS) 6841(1987);

- Dynamic Response Index (DRI)(Payne, 1975); and

- Air Standards Coordinating Committee (ASCC, 1982);

Most standards cited in MIL-STD 1472D are predicated on the ISO 2631 "Guide for the Evaluation of Human Exposure to Whole-Body Mechanical Vibration" (ISO 2631, 1982). The ISO standard, which is largely based on subjective measurements of fatigue and comfort rather than health, does not adequately account for the health effects of repeated shock (Village and Morrison, 1989).

It is essential that cause-effect relationships between the mechanical environment and injury (acute and chronic) be determined for quantification of health effects. Thus, the Army Surgeon General urgently required the Medical Research and Development Command to develop exposure standards for repetitive whole-body shocks which are relevant to the environment of soldiers operating modern tactical ground vehicles and weapon systems.

Military significance

New TGVs developed by the U.S. Army are generally lighter in weight and capable of considerably higher speeds than their predecessors. This combination of lower weight and higher speed over rough terrain produces repetitive mechanical shocks that are transmitted to the soldier primarily through the seating

system. Under certain operating conditions, exposure to shock and vibration poses health and safety threats to the crew and performance degradation due to fatigue (Larson, Wells, and Kaplan, 1973; Heslegrave et al., 1990). Anecdotal evidence indicated that 50 percent of a company reported blood in the urine following operation of fast attack vehicles (USAARL, unpublished).

Health hazard assessment program

The U.S. Army established a Health Hazard Assessment (HHA) Program (AR 40-10) to evaluate and control health hazards in support of the Army's military capabilities and performance. Overall, the HHA Program is an integrated effort that supports all areas and mission needs. Its specific objectives which are relative to this contract are: to preserve and protect the health of individual soldiers; to enhance soldier performance; to reduce readiness deficiencies related to health hazards; and to reduce personnel compensation claims by eliminating or reducing injury or illness caused by health hazards associated with the use of Army systems (Liebrecht, 1990).

Health hazard assessment refers to the process of identifying, evaluating, and controlling risks to the health and effectiveness of personnel who test, use, service, or support Army systems. Many health hazard effects are not immediate and may appear only after months or years of exposure. Such delayed effects may limit long-term contributions to the Army and may develop into serious health problems in the future, although the short-term impact on the soldier's performance may be minimal (Liebrecht, 1990).

The HHA program utilizes resources to apply biomedical knowledge and principles to support the development of military material systems (Liebrecht, 1990). In relation to this project, soldiers who operate or are transported in TGVs are exposed to mechanical forces which are considered health hazards, including vibration and shocks.

Overview of study design

The overall objective of the project was to develop a dose-effect model that will predict, and ultimately minimize, the risk

2

of injury to a soldier when exposed to the repeated shock environment of tactical ground vehicles. The project, which spanned six years and five phases, was carried out between July, 1991 and July, 1997. The five phases and the time frame in which they were conducted are listed in Table 1.

Table 1
Study design

Phase	Title	Dates
Phase 1	Literature Review	July, 1991 to July, 1992
Phase 2	Characterization of the Environment	July, 1991 to February, 1993
Phase 3	Pilot Studies	July, 1992 to July, 1993
Phase 4	Experimental Phase	July, 1993 to July, 1995
Phase 5	Recommendations for a Health Hazard Assessment Method	July, 1995 to July, 1997

In Phase 1, a review of literature was conducted. This phase concluded with a list of potential measures or indices that might be sensitive to shock and impact, and that could be evaluated in the pilot experiments in Phase 3.

Phase 2, the vehicle characterization phase, ran concurrently with Phase 1. Phase 2 involved receiving and analyzing tapes of data from the United States Army Aeromedical Research Laboratory (USAARL) containing acceleration measurements from TGVs. A variety of unique characterization methods were developed and programmed for data containing mechanical shocks and repeated impacts. These methods were meant to be more sensitive to shocks than previously available methods. This allowed "typical" vibration and repeated shock environments to be identified and defined based upon the TGV data tapes. The characterization methods were used to develop motion signatures to drive the multi-axis ride simulator (MARS) for Phase 3 and Phase 4 experiments.

Phase 3 consisted of pilot tests conducted using the MARS facility at USAARL, Fort Rucker, Alabama. In this phase, a number of biomechanical, physiological and biochemical indices were measured in short duration (6 minute) and longer duration (1 and 2 hour) experiments.

Phase 4 was the full experimentation phase. A series of six short and long term experiments were conducted at the MARS between August 1994 and January 1995. Details of the experimental methods, data analysis and results were provided in the Phase 4 report. Phase 5 was focused on the development of a model of health hazard assessment of mechanical shock and repeated impact in Army vehicles. The HHA method allows for the evaluation of repeated mechanical shock exposure and assignment of a Risk Assessment Code (RAC) according to the Army HHA program (AR 40-10).

Human subject use justification

The primary objective of this project was to evaluate the human response to mechanical shocks and repeated impact with the intention of developing a health hazard index for exposure to motion in army vehicles. Thus, in order to study the human response to mechanical shocks and repeated impact, the use of human subjects was an absolute requirement. Other species do not have the same biomechanical structure, transmission characteristics, physiological or biochemical response to mechanical shock.

Human use and ethics review

All studies complied with USAARL Policy No. 70-3 and Regulation 70-25 in conducting research with human subjects, including the necessity for informed consent, based on the accurate presentation of this protocol, and the right of withdrawal of the subjects. For the protection of human subjects, the investigator(s) have adhered to policies of applicable Federal Law 45 CFR 46.

The study protocols were approved for individuals by the Ethics Committee at the University of British Columbia or at Simon Fraser University as well as the Human Use Committee at USAARL and the U.S. Army Medical Research Development Command

(USAMRDC). All subjects completed and signed informed consent forms and voluntary affidavit forms before participating in the experiments.

Selection of subjects

Subjects were recruited from U.S. Army personnel assigned to Fort Rucker. Subjects were recruited through a notice published in the USAARL newsletter and personal communication. Selection criteria were detailed in experimental protocols developed for the Human Use Review Committee. Some subjects who did not satisfy all exclusion criteria were eliminated. All were required to have experience with motion, either in TGVs or air transport. Ten subjects participated in Phase 3 experiments and fifty four subjects participated in Phase 4 experiments.

Only male subjects were included in this study. A male subject pool was initially selected for this project based on the restricted number of subjects in the experimental design and because at the outset of this study females did not participate in combat maneuvers in TGVs. Thus, earlier phases of this study focused exclusively on male subjects as applied to crew members of tactical combat vehicles. A change in the design of the project to include females at Phase 4 would have adversely affected the experimental design, as female subjects are likely to have a different response to shock and impact than males, based on differences in body morphology and hormonal environment. Hence, to maintain consistency with other phases of the project and reduce variability in the data, only male volunteers, ranging from 19 to 40 years of age, were recruited.

Subject briefing and informed consent

Each subject completed the Volunteer Registry Data Sheet (USAMRDC Form 60-R) which documents participation in research conducted or sponsored by U.S. Army Medical Research and Development Command. These forms were copied and filed with the appropriate governing bodies to allow any delayed effect from these experimental protocols to be traced through USAMRDC. A verbal explanation of the experimental protocol was given to each subject prior to motion exposure.

Medical screening

Each subject completed a medical questionnaire to draw special attention to specific disorders and conditions as specified in the British Standards Institute 7085: 1989, "Safety aspects of experiments in which people are exposed to mechanical vibration and shock" and in the "Guide to experimentation involving human subjects", Human Experimentation Safety and Ethics Committee, Institute of Sound and Vibration Research, Southampton University, U.K. Prior to participation in experiments, subjects underwent a medical examination conducted at USAARL, Fort Rucker by the medical monitor of this project (a USAARL physician).

Orientation

Prior to collection of experimental data, each subject participated in at least a 15 minute trial exposure on the MARS to familiarize him with the motion environment. The exposure consisted of three five minute exposures to 2 g shocks delivered at a rate of 32 per minute in each direction (i.e., positive and negative) of the x, y, and z axes.

Shock and impact

When subjects are exposed to motion environments, there are two distinct effects of motion input at the seat.

1. The direct effect of shock (transmission), and

2. The indirect effect of secondary impact (of the human with the seat).

Griffin (1990) defines shock as a sudden change in force, position, velocity or acceleration that excites transient disturbances in a system. In this report, mechanical shocks are low frequency (2 to 20 Hz) events imparted by direct transmission of vehicle motion through the seat.

An impact is defined as a single collision between one mass and a second mass (Griffin, 1990). The high frequency event (20 to 150 Hz) resulting from the collision of the subject with

the seat is an impact. The biomechanical events associated with these impacts are not clearly understood.

Waveform frequency

Throughout this report, shock waveform frequency is defined as the inverse of the time period of the biphasic impact waveform and where the shock waveform is presented as a damped sinusoid consisting of a single time period.

Limitations inherent in the study design

Certain limitations which were identified at the outset of this project presented a challenge with respect to development of the experimental design and achieving the final deliverable. Throughout the study, extrapolation of short-term exposure effects to chronic health effects remained the most difficult issue to overcome. Limitations in the experimental design which were identified at the proposal stage included:

- extrapolating short-term exposure effects to chronic health effects;

- detecting significant health-related responses at low vibration levels;

- ethical concerns of exposing subjects to risk of injury since short term health effects may only be observable at high shock severity;

- because of the above ethical constraint, the need to conduct the investigation at exposure levels below that likely to cause serious health effects;

- developing a model based on acute experimental data;

- needing to link shock signatures for the vehicles and environments in which they are driven directly with chronic health data that have been reported in the epidemiological literature;

- limiting the number of subjects that can be tested within the scope of the project relative to the large sample

7

population required to validate sensitivity contours (Oborne, 1983); and

- determining the frequency range of motion in TGVs and incorporating similar frequency spectra in the motion exposure experiments.

As the study progressed, other limitations were apparent with particular respect to the analysis, interpretation and application of the data. These are discussed following the summary of Phase 5.

Risk assessment and safety procedures

Risk assessment

The quantity of vibration and shock which was included in the motion signatures of each experiment was compared to appropriate standards: British Standard "Measurement and evaluation of human exposure to whole-body mechanical vibration and repeated shock" BS 6841 (1987); and Air Standardization Coordinating Committee "Human tolerance to repeated shock" ASCC Advisory Publication 61/25 (1982). The International Organization for Standardization "Guide for Exposure of Human Response to Whole Body Vibration" ISO 2631 (1982), although widely used, is not applicable to exposures containing non-stationary events such as repeated shocks. It was therefore rejected for this purpose.

Appendix A of the British Standard 6841 is designed to account for the effects of repeated shocks, and as such is a more appropriate basis for assessment of the exposures contained in this study. Hence, the various motion signatures that were developed in this project were designed to produce specific vibration dose values (VDVs) as defined in the BS 6841. However, the BS 6841 does not specify limits of comfort or safe exposure, as it is considered that there is insufficient data on which to base these limits.

A primary concern in this project was the safe level of acute exposure measured during long duration experiments. For this reason we also referred to ASCC 61/25 (1982) for guidance. The ASCC guidelines indicate the magnitudes and numbers of shocks (in the +z direction) that can safely be

8

sustained during a 24 hour period. The guidelines include levels of moderate discomfort, severe discomfort and 5% injury risk.

Safety procedures

A number of operating procedures were incorporated into the experimental protocol to ensure the safety of the subject and research team. These included subject screening, limiting shock exposure dose, controls in the experimental design, and safety features built into the MARS facility.

Safety in experimental design

The experimental design controlled shock exposure dose based on guidelines of BS 6841, (1987) ASCC Advisory Publication 61/25 (1982) and ISO 2631. The cumulative exposure to motion signatures of any one subject did not exceed 20 hours per week, or 30 hours per month. The maximum daily exposure took place in Phase 4, experiment LT3 in which each subject was exposed to motion for a maximum of 7 hours in one day. The maximum weekly exposure took place in Phase 4, experiment LT4, when each subject was exposed to the motion for a maximum of 4 hours per day for 5 consecutive days. Subjects did not participate in any further experiments for a minimum of one month after participation in either of these experimental conditions.

Report structure

Appendix A lists the team members who contributed to this project. A brief biography of the primary team members is provided in Appendix B. Figures are illustrated in Appendix C. A list of the major equipment that was used is provided in Appendix D. Appendix E lists the publications that resulted from this contract. The references cited in this report are provided in Appendix F, followed by a glossary of terms in Appendix G.

Phase 1: Literature review

Phase 1 introduction

A thorough review of the literature was conducted in Phase 1 of this study. A detailed report was submitted as the Phase 1 report and has subsequently been published (Village et al., 1995a). An annotated bibliography of more than 1,200 articles was also provided to USAARL at the completion of this phase. Since completion of Phase 1 in July, 1992, several relevant articles have been published that were not included in this review.

Databases that were searched included: Aerospace Database; Biosis; Embase; EI Compendex Plus; Inspec; Mathsci; Medline; NIOSH - Occupational Safety and Health; Pascal; NTIS; and Academic Library Catalogues - University of British Columbia, Simon Fraser University. Articles were obtained through the following document suppliers: Aerospace Medical Research Laboratories, Wright Patterson Air Force Base; Acoustical Society of America; American Institute of Aeronautics & Astronautics; British Library Document Supply Centre; Canadian Institute for Scientific and Technical Information; Canadian Standards Association; Defense Technical Information Center; European Rail Research Institute; Inspec; Institute of Sound and Vibration Research at University of Southampton; International Commission on Occupational Health; Motor Industry Research Association; National Technical Information Service; Society of Automotive Engineers; Standards Council of Canada; National Aeronautics and Space Administration; Royal Aircraft Establishment Farnborough; Simon Fraser University Library; and University of British Columbia Library.

Phase 1 objectives

The objectives of the literature review were:

- to review the known health effects of vibration and shock;

- to examine the dynamic response of seated humans to vibration and mechanical shock;

- to evaluate existing models, standards and guidelines for exposure to vibration and shock;

- to review mathematical methods to quantify the complex acceleration wave in vehicle motion signatures; and

- based on existing knowledge, to recommend the best measurement indices for HHA to be included in the pilot experiments.

Topic areas of the literature review

Topic areas which were covered in the literature review are listed in Table 2. In the Phase 1 report, conclusions were provided at the end of each topic area. Where relevant, tables were constructed to facilitate comparison of results from different papers. A summary of the conclusions, and a discussion of the relevance of the literature to development of a standard for the HHA of mechanical shock and repeated impact are provided below.

Table 2
Topic areas of literature review

Health effects of vibration: Epidemiological research

Subjective response to mechanical vibration and shock

Physiological effects of vibration and repeated shock

Biochemical effects related to mechanical shock and repeated impact

Muscle response to vibration

Biodynamic response: Transmission, impedance and apparent mass

Biomechanics

Biodynamic models

Standards and guidelines for shock and vibration

Vibration data collected in the field

Signal processing

Health effects of vibration: Epidemiological research

The epidemiological literature was reviewed in the following areas:

- heavy and miscellaneous equipment operators: back pain;

- heavy equipment operators: back disorders;

- pilots: back pain;

- pilots: back disorders;

- other health problems;

- relationship between WBV and health disorders; and

- possible etiologies of health disorders due to WBV.

Knowledge of the health effects of exposure to WBV has been obtained through a large number of studies of disparate groups exposed to vehicle accelerations (for review, see Griffin, 1990). Health effects have been classified as either acute or chronic. Acute effects exhibit their maximum response almost instantly and are therefore associated with discrete events. An example of an acute injury is spinal fracture as a result of a single vertical shock during aircraft ejection. Chronic effects develop over time and are usually associated with cumulative exposures. Epidemiological studies to determine chronic effects were largely based on retrospective or cross-sectional surveys. Others investigated objective findings of disease through medical records or radiological procedures.

Several of the epidemiological studies evaluated the effect of chronic exposure to vibration and shocks in heavy equipment operators from industries such as agriculture, construction, mining, forestry, and the military (Rosegger and Rosegger, 1960; Konda et al., 1985; Beevis and Forshaw, 1985; Boshuizen, Bongers, and Hulshof, 1990; and Milby and Spear, 1974). Some studies focused on subjective symptoms of health problems (e.g., backache), while others investigated objective findings of

disease, such as back disorders as diagnosed through clinical or radiological findings (e.g., intervertebral disc herniation and spondylolisthesis).

Most of the disorders described in relation to human exposure to vibration are not specific to vibration, but occur generally in the population. The most common health problems are back pain and disorders (such as damage of the intervertebral disc, and degeneration of the spinal vertebrae). To a lesser extent there are reports of gastrointestinal disorders, abdominal pain, increased urinary frequency, prostatitis, hemorrhoids, hypertension and cardiac disorders. Each can be associated with or aggravated by other ergonomic or environmental problems (Dupuis and Zerlett, 1986).

Although some of the epidemiological studies had appropriate control groups and quantified the magnitude and duration of vibration exposure, few satisfied the criteria for determining cause-effect relationships between vibration exposure and possible chronic health effects. Many studies were also confounded by workers leaving occupations involving exposure to vibration with advancing age or onset of disease, resulting in biased population data. Nevertheless, similar findings have been described (Dupuis and Zerlett, 1986; Hulshof and van Zanten, 1987; Seidel and Heide, 1986).

Despite the fact that few studies satisfied the criteria for determining a cause-effect relationship, and a clearly defined dose-response could not be determined between vibration and health effects, there was sufficient data to support the following conclusions.

- Long-term exposure to vibration can be harmful to the spine and possibly other organs of the body (e.g., gastrointestinal and cardiorespiratory systems).

- No single disorder can be linked solely to vibration or shock.

- Rather than causing specific pathologies, it appears that vibration accelerates the onset of currently recognizable syndromes.

Although some studies included exposure to both vibration and repeated shock, few studies included repeated shock as a variable for consideration. None have quantified accurately the magnitude and duration of repeated shock.

Subjective response to mechanical vibration and shock

A review of the literature concerning subjective response to vibration, mechanical shock and repeated impacts was important for several reasons. Many researchers have characterized comfortable or acceptable levels of vibration and shock by using subjective measures in a wide range of conditions and types of vibration, in controlled laboratory studies with large numbers of subjects. Descriptions of sensations at the limit of subjective tolerance provide useful information about the possible mechanical and physiological response of the body. Finally, the international standards which govern exposure to industrial vibration (e.g., ISO 2631) are based largely on data from subjective comfort studies. Topic areas that were reviewed in this area included:

- limitations of subjective data;

- definition of comfort;

- techniques for measuring comfort;

- subjective response to sinusoidal vertical (a_z) vibration;

- subjective response to sinusoidal horizontal (a_x and a_y) vibration;

- subjective response to random vibration;

- subjective response to multiple directions of vibration;

- effect of exposure duration on discomfort;

- subjective response to rotational vibration;

- ride comfort models;

- discomfort from shocks and impulsive vibration;

- tolerance to vibration and shock; and

- subjective effects of noise in combination with vibration.

Frequencies at which maximum discomfort is observed have been defined for the three translational and rotational axes. Compared to exposure in a single direction, subjects are usually

14

more sensitive to random, rather than sinusoidal vibration, and to vibration exposure in multiple directions. The increased discomfort imposed by additional frequencies or directions is best predicted by the root sum of squares method (Fairley and Griffin, 1988; Griffin and Whitham, 1977; Mistrot, Donati, and Galmiche, 1990). There seems to be little evidence to support an increasing level of discomfort with increasing duration of exposure (except for exposures of a few minutes).

Ride comfort models derived from field and laboratory studies have generally concluded that vibration levels between 0.6 and 0.8 m·s^{-2} are on the border of being described as uncomfortable. However, one's definition of comfort depends on the environment. The location of the axis of rotation is important in determining subjective comfort of rotational motion, as subjects are more sensitive to pitch and roll vibration than yaw. For a seated individual, vibration of the seatback, and to a lesser extent the feet, can also be important factors in determining discomfort.

There have been fewer investigations of subjective response to shock or repeated impact. One important drawback in the literature is that there is no universally accepted definition of shock and impact. Exposure to random vibration with superimposed shocks tends to be more uncomfortable than random vibration alone.

A number of studies have attempted to determine which method best characterizes vibration with repeated shocks. Exponents of two and four (the root mean squared - rms; and the root mean quad - rmq) seem equally associated with discomfort. (In Phase 2, exponents up to 12 were employed to analyze the seat motion data and to characterize the motion signatures). The exponent of best fit depends upon the acceleration time history of the motion, including separation time between shocks, the range of signals presented to the subjects, background vibration levels, frequencies of the shocks, anticipation of shocks and a subject's experience with the motion.

The limits of subjective tolerance to vibration and shocks have been derived from subjective comfort studies, accidents such as aircraft escape and free falls from lifeboats, and mathematical models. Generally, at lower frequencies (4 to 8 Hz) the centrally located organ-tissue systems are most affected; the peripherally regions become affected to a greater extent

15

as frequency is increased. Tolerance to shocks depends largely on the acceleration time history.

Descriptions of sensations at the limit of tolerance provide useful information about the possible mechanical and physiological responses of the body. Explanations were described by Forshaw and Ries (1986) for common symptoms related to tolerance limits which are listed in Table 3.

Table 3
Subjective symptoms reported at the limit of tolerance

Abdominal pain	stretching and deformation of the terminal ileum, cecum, hepatic flexure and transverse colon.
Chest pain	stretching of major vessels originating at he base of the heart and mechanical stimulation of diaphragmatic pericardium.
Testicular pain	displacement of the spermatic cord and deformation of the testicles.
Head symptoms	displacement of facial skin and subcutaneous tissues about underlying bony structures.
Dyspnea	alternating displacements of thoraco-abdominal system and pulmonary hemodynamics such as pooling of blood in pulmonary vessels resulting in pulmonary congestion.
Anxiety	pain, stimulation of proprioceptive system and respiratory impairment.

For a number of reasons, studies investigating subjective response have reported variable results. Lower levels of vibration and repeated shock may contribute to chronic health problems, or an earlier onset of problems, but may not seem subjectively uncomfortable to the persons exposed. However, there is no evidence in the literature to support a time dependency of long-term reduced tolerance. Some studies have also concluded that subjective response may not be an effective way to discriminate the effects due to repeated shock.

Hence, it is difficult to link subjective results to current objective efforts to describe the health hazard effects of vibration and shock. Despite the limitations, studies which report subjective data provide an important adjunct to objective health findings since subjective comfort is a direct response to mechanical sensations within the body.

Physiological effects of vibration and repeated shock

Vibration and shocks produce acute effects on a number of systems in the body. Numerous reviews are available (Barnes, 1987; Guignard, 1972a,b, 1974, 1985; Ramsay and Beshir, 1981; Weaver, 1979). In this project, the literature concerning physiological effects of vibration and repeated shock was divided into the following categories:

- general;

- cardiovascular;

- respiratory;

- gastrointestinal;

- combined effects of noise and vibration; and

- effect of mental workload.

There has been little work on the effect of repeated shocks on physiological parameters. Most of the information reviewed has come from literature on the acute effects of WBV on animals and humans. A significant problem is the extrapolation from acute physiological effects to long-term health hazards.

Physiological effects of vibration and repeated shocks are related to two potential mechanisms: the movement of organs and tissues; and a generalized stress response related to intensity and duration of exposure. Many of the responses to vibration are attributed to stimulation or over-activation of the sympathetic nervous system. This can result in increased concentrations of catecholamines and vasoactive metabolites which in turn cause a generalized stress response.

Some of the cardiovascular and respiratory responses to WBV mimic the effects of moderate exercise. For example, an increase in heart rate, cardiac output, respiration rate and oxygen uptake occur in response to WBV. In some cases, peripheral

17

vasoconstriction has also been reported (Abu-Lisan, 1979; Spaul, Spear, and Greenleaf, 1986). Acute pathological effects of vibration and shock include injury to viscera, lung and myocardium (Guignard, 1972b), bleeding in the gastrointestinal system (Sturges et al.,1974), and occasionally, hemorrhage of kidney and brain (Guignard, 1972b). It is possible that mechanical vibration of the intestines will increase motility, or movement of ingested material without appropriate breakdown or absorption taking place. A number of epidemiology papers have also suggested that hearing loss due to noise is exacerbated by vibration (Chernyuk and Tashker, 1989; Rehm and Wieth, 1984).

Biochemical effects related to mechanical shock and repeated impact

Biochemical measures in blood and urine are routinely measured in a clinical setting to evaluate stress and strain on the human body. These tests are used to detect physiological and metabolic abnormalities, as well as tissue or organ damage. Careful interpretation of biochemical data can often differentiate between acute and chronic dysfunction.

Because of the limited number of studies reporting biochemical effects in humans exposed to vibration or shock, animal research and exercise physiology literature was reviewed to identify a biochemical marker for:

- general stress;

- fatigue; and

- tissue or organ damage.

Physiological and anatomical differences between humans and animals with respect to size, resonant frequency of internal organs, and the response of a quadruped compared with a human in a seated posture limit direct comparison of data from animal and human experiments. Conclusions from animal studies also have to be interpreted with caution because in some instances, the level of vibration used in animal experiments was extremely high compared to what a human would be exposed in a transport vehicle.

The biochemical indicators which were considered for inclusion in Phase 3 pilot studies are listed in Table 4, with an indication of their potential relevance in terms of tissue damage or physiological disturbance.

18

Table 4
Biochemical indicators in biological specimens

Clinical Relevance	Metabolite	Biological Specimen
Bone remodeling/ Joint Damage	Alkaline Phosphatase	Blood
	Hydroxyproline	Urine
Capillary endothelial damage	von Willebrand's Factor Antigen	Blood
Electrolyte shift	Calcium Magnesium Potassium Sodium	Blood
Fatigue	Ammonia Lactate	Blood
Fluid Shift/ Dehydration/ Protein Shift	Hematocrit Hemoglobin	Blood
	Specific Gravity	Urine
Gastrointestinal Injury	Hemoglobin	Feces
Hypoglycemia	Glucose	Blood
Hyperuricemia (1° or 2°)	Uric Acid	Blood
Inflammation/ infection	White blood cell profile	Blood
	WBCs Bacteria	Urine

Table 4 cont'd
Biochemical indicators in biological specimens

Clinical Relevance	Metabolite	Biological Specimen
Kidney/liver/urinary tract function	Blood Urea Nitrogen Creatinine	Blood
	Casts Crystals Mucous Protein RBCs	Urine
RBC damage	Bilirubin Free Hemoglobin Haptoglobin	Blood
	Hemoglobin	Urine
RBC vs. liver dysfunction	Total vs Conjugated Bilirubin	Blood
Skeletal muscle damage	CPK LDH	Blood
Skeletal muscle damage	Myoglobin	Blood/Urine
Skeletal vs cardiac muscle damage	CPK and LDH (isoenzyme profile)	Blood
Stress/Adrenal Function	Cortisol Catecholamines	Blood

Biochemical responses indicating fatigue have been measured in the vibration literature, but are more commonly reported in relation to physical exercise. A biochemical marker of fatigue would be an important factor in a health hazard index, since increased fatigue (monitored as a decrease in metabolic potential, or a decrease in force generated by muscle) will ultimately result in decreased physical and cognitive performance, and possibly increased recovery time. Peripheral muscle fatigue has been related to changes in carbohydrate metabolism (lactate, glucose), protein and energy metabolism (ammonia), cortisol, and electrolyte balance (e.g., potassium, magnesium and calcium) (Roberts and Smith, 1989).

Elevation of serum lactate concentration following physical exercise, and presumably following vibration exposure, depends on the intensity and duration of the stressor, individual fitness, fatigue, and muscle fiber composition (Farber et al., 1991; Kraemer and Brown, 1986; Long et al., 1990). Serum lactate is elevated, relative to controls, following nap-of-the-earth helicopter flights. Anderson et al. (1977) interpreted this to reflect increased muscular strain. Kamenskii and Nosova (1989) reported an elevated lactate concentration, immediately following WBV exposure simulated to represent modern transport cabs.

While prolonged exercise provides a useful model of hypoglycemia, the effect of vibration exposure on blood glucose concentration is of greater interest in the present study. Maintenance of blood glucose is particularly important in an occupational setting because hypoglycemia can interfere with task performance. A single four hour exposure to low amplitude, high frequency WBV in rabbits and in dogs results in a decrease in blood glucose and glycogen (Sinitsyn, Rumyantsev, and Voronova, 1964). The observed decrease is more pronounced following repeated exposure to vibration. In contrast, Dolkas, Leon and Chackerian (1971) reported an increase in plasma glucose in rats following short term exposure to WBV (4.7 Hz, 17 $m \cdot s \cdot^{-2}$ for 10 min.). Because of the great difference in vibration exposure, it is difficult to compare these results. No studies were reviewed which measured the effect of vibration and shock exposure on blood glucose in human subjects.

The pituitary-adrenocortical system plays a major role in maintaining biological homeostasis in response to various stimuli. Prolonged elevation of cortisol, in the absence of a stressor, is a reflection of chronic adrenal stress, or over activation. Both urinary and plasma adrenal steroid

21

metabolism is altered following WBV exposure in humans (Litta-Modignani et al., 1964; Blivaiss, Magid, and Litta-Modignani, 1964) and in rats (Dolkas, Leon, and Chackerian, 1971; Ariizumi and Okada, 1983).

There are only a few reports in the literature on the effect of vibration exposure on serum electrolytes. Prolonged repeated exposure to WBV (12 Hz, 15 m$\cdot$s^{-2}, 5 hr$\cdot$day^{-1}, 130 hr) has been reported to result in increased serum potassium in monkeys (Badger et al., 1974). This study did not relate the change in serum potassium to any physiological parameters.

Kosmakos, Keller, and Collins (1975) found that serum magnesium (Mg^{2+}) concentration in rats was unchanged immediately following 4 minutes vibration at 3 frequency ranges from 12 to 211 Hz, although Mg^{2+} was consistently lower 1 day following vibration treatment. While it is difficult to extrapolate these data to prolonged WBV, a measure of serum magnesium concentration, in conjunction with EMG data, could be used as another indication of muscular fatigue.

During physical exercise, there is normally little change in serum calcium ion (Ca^{2+}) concentration (Long et al., 1990). A change in Ca^{2+} concentration has been reported, however, in response to vibration exposure. Badger (1974) observed a reduction in serum Ca^{2+} concentration in monkeys following prolonged vibration exposure (12 Hz, 15 m$\cdot$s^{-2}, 5 hr$\cdot$day^{-1}, up to 130 hr exposure). Although no explanation is given, it could be related to a mechanism of muscle fatigue. Elevated intracellular calcium is also important in the etiology of local skeletal muscle damage (Jackson, Jones, and Edwards, 1984; Duncan and Jackson, 1987; Duncan, 1987; Duncan, 1988), and subsequent release of skeletal enzymes (lactate dehydrogenase-LDH, creatine phosphokinase-CPK) to the circulation (Jackson, Jones, and Edwards, 1984).

While Raynaud's phenomenon is not specific to individuals who have been exposed to vibration (primarily hand-arm or segmental vibration), an inflammatory response has been linked to Raynaud's phenomenon both of occupational and non-occupational origin (Langauer-Lewowicka, 1976). Individuals who have Raynaud's phenomenon experience finger blanching on cooling.

Animal studies have shown that exposure to vibration results in damage to heart, lung, brain, kidney, gastrointestinal (GI) tract, liver, skeletal muscle, adrenal glands, and reproductive

organs (Aria, Onozawa, and Iwata, 1990; Badger et al., 1974; Boorstin, Hayes, and Goldman, 1966; Cope and Polis, 1959; Inaba and Okada, 1988; Ivanovich, Antov, and Kazakova, 1977; Ivanovich, Antov, and Kazakova, 1981; Mandel, Robinson, and Luce, 1962; Megel et al., 1962; Megel et al., 1963; Sackler and Weltman, 1966). Some damage is detectable in blood and urine, while others require histological examination of tissue. Studies involving humans cannot directly assess internal organ damage, although evidence of internal damage is inferred by data reported in the epidemiological literature. In some instances, biochemical markers in blood, urine, and feces have been used as evidence of altered internal organ function or actual physical damage.

Jayson et al. (1991) proposed that low back pain associated with exposure to WBV may be due to damage to the interior walls of blood vessels (the vascular endothelial lining). In one human study, the concentration of von Willebrand's Factor (vWF) antigen in serum is significantly increased after 25 minutes of exposure to vibration at 5 Hz (which is the ISO 2631 Fatigue Decreased Proficiency Limit). Serum vWF is elevated in patients with Raynaud's syndrome (Ikehata et al., 1980; Cimminiello et al., 1991). Von Willebrand's Factor is frequently used to assess vascular damage in clinical settings, ranging from systemic vasculitis to diabetic microangiopathy (Pasi et al., 1990; Bleil et al., 1991; Castillo et al., 1991; Porta, La Selva, and Molinatti, 1991). If vascular damage occurs in response to WBV, measurement of von Willebrand's Factor would be a useful marker for the HHA method.

In humans, occupational exposure to vibration, mechanical shock and impact is often linked with bone and joint dysfunction, particularly in the spine (Rosegger and Rosegger, 1960; Guignard, 1972a; Radin et al., 1973; Westgaard et al., 1986; Froom et al., 1986; Guidotti and Cottle, 1987; Sandover, 1988). Damage to connective tissue in joints can be detected by an increase in hydroxyproline (a chemical found almost exclusively in collagen of connective tissue) excretion in urine. Kasamatsu et al. (1982) observed a significantly greater urinary excretion of hydroxyproline in workers exposed to occupational hand-arm vibration. If joint injury is suspected as a result of whole-body exposure to vibration, urinary excretion of hydroxyproline may be a useful indicator.

Evidence of gastro-intestinal (GI) tract lesions may be detected by the presence of fecal hemoglobin. Blood in feces

23

has been observed in monkeys exposed to high levels of vibration (12 Hz, 15 m·s^{-2}, 5 hr. daily, for a total of 130 hr.). Within 2 days of initiating vibration exposure, tests were positive for blood in stools (Badger et al., 1974; Sturges et al., 1974).

Megel et al. (1963) suggested that local hypoxia is a major contributor to internal organ damage during vibration stress in rats. An increased frequency of injury to internal organs (including GI tract and stomach) as a result of exposure to high frequency, low amplitude vibration is observed at altitudes greater than 10,000 feet. The greater incidence of injury at altitude is attributed to a reduction in the partial pressure of oxygen which increases the local hypoxia created by a reduction in blood flow to internal organs (Megel et al., 1963). It is possible that vibration and shock may increase the incidence of GI bleeding when blood flow to the internal organs is already reduced.

Damage to liver by vibration exposure is implied in studies reporting increased serum activity of liver enzymes (Cope and Polis, 1959; Mandel, Robinson, and Luce, 1962). Tambovtseva (1968) also reports chronic changes reflecting liver damage (i.e., changes in serum protein content, protein fractions, and albumin/globulin ratios) in excavator operators exposed to occupational vibration for a minimum of 5 years.

The goal of the HHA method is to reduce exposure to vibration and repeated shock below a level that may cause physical damage or disability. The inclusion of biochemical tests which would indicate fatigue, stress, or tissue damage were recommended for Phase 3 and Phase 4 experimental studies. The intention was to identify a biochemical marker for the development of the HHA method that would provide an objective index for health assessment. Biochemical evidence of severe physiological disturbance, and tissue or organ damage, would confirm that a "safe" exposure limit has been exceeded.

Muscle response to vibration

The response of paraspinal muscles to WBV has been studied using electromyography (EMG) to assess localized muscle fatigue, phase and timing relationships between muscle response and acceleration, and to estimate compressive loading and torque about the spine. These parameters were of interest in the development of the experimental protocol because of their association with stabilization of the body during motion and

24

because of possible association with back pain and injury to spinal tissues.

Muscle fatigue may diminish the ability of muscle to adequately compensate for perturbing forces, while out-of-phase or untimely muscle response can contribute to postural destabilization and increase both torque and compressive loading of the spine (Seroussi, Wilder, and Pope, 1989; Seroussi et al., 1987).

The literature review focused on the following areas:

- EMG methods (signal conditioning, motion artifact and noise);

- muscle fatigue;

- EMG markers of localized muscle fatigue;

- paraspinal EMG fatigue parameters and back pain;

- WBV and paraspinal muscle fatigue;

- timing of the muscle response to vibration;

- muscle activity and forces at the spine; and

- muscle response to shocks.

Several factors affecting the usefulness and limitations of EMG with respect to the development of the HHA were outlined.

- The contribution of muscle tension to forces on the spine can be estimated using EMG. However, the precision of this estimate is limited by the difficulty in adequately calibrating an EMG-force relationship for a dynamic process, and by the complex phase relationship between acceleration and muscle response.

- The relative timing of tension generation by muscle is important for an effective homeostatic response to vibration and shocks. However, estimation of the timing of muscle tension development from EMG requires incorporation of an electro-mechanical delay between onset of myoelectric activity and tension production. This delay is influenced by the dynamic characteristics of the muscle, and can only be roughly estimated.

25

- Fatigue of paraspinal muscles can be quantified through various analytical techniques. In spite of this, the significance of any measured fatigue can only be inferred with respect to back pain and a diminished capacity for postural control. The reliability and repeatability of many of the analytical techniques is poor.

- No inference of tissue damage can be drawn directly from EMG data. Complementary analysis of biochemical markers for tissue damage may allow the establishment of an association between EMG fatigue parameters and tissue damage.

- The ability of the musculature to respond to successive shocks can be estimated using a recovery rate model. The method of defining the steady state response to background WBV, however, has not been established. The use of phase space trajectories may provide a method of quantifying steady state recovery.

Biodynamic response: transmission, impedance and apparent mass

The biodynamic response to exposure to vibration and shocks was reviewed to determine how the displacement of tissues and the forces transmitted to them may be altered as a function of time. Specific topic areas which were reviewed included:

- transmission;

- transmission from the seat to the head;

- thoracic abdominal displacement;

- accelerations of the spine;

- vibration and shocks in normal activity;

- impedance; and

- apparent mass.

Despite an abundance of information on transmission, impedance and apparent mass, the dynamic response of individual body segments to vibration and shocks remains imperfectly defined. The uncertainty arises due to the strong coupling action of adjacent body structures demonstrated in experimental results, which confounds the assignment of mechanical properties to specific structures.

26

Sandover (1982) identified a resonance frequency of 5 Hz that was attributed to the spinal column and pelvis. However, the intervertebral discs are too stiff in axial compression to attenuate low frequency shocks (Markolf, 1970; Belytschko and Privitzer, 1978; Smeathers, 1989). Thus, the resonance frequency of 5 Hz attributed to the spine is likely derived from the combined properties of upper torso mass, flexibility of the spinal column, and the stiffness and damping of the supporting muscles and ligaments. Belytschko and Privitzer (1978) concluded that the resonance of driving point impedance shown at 5 Hz resulted from a combination of pelvic, visceral and spinal elements, and reflected the elastic properties of the buttocks, abdominal wall and spinal flexion respectively.

Although measures of driving point impedance and apparent mass provide useful indications of the response of the body, these measures do not provide sufficient detail to determine the behavior of, or stresses acting on, individual systems such as the abdomen or spine. In particular, there is a lack of information regarding the non-linearity of individual systems, particularly in response to shocks.

Biomechanics

Epidemiological studies which were reviewed pointed towards a fundamental difference between the chronic effect of exposure to WBV compared to repeated shock. Low back pain and injury due to accelerated degeneration of the spinal unit were described as a hazard of chronic vibration exposure, whereas impact injuries involved fractures of the vertebrae.

Mechanical systems fracture under severe loading and suffer fatigue failure in response to low level vibration. The literature supported an obvious analogy between the effects of mechanical shock and vibration on living systems and the failure modes of engineering materials. It followed that any attempt to gain a quantitative understanding of the physical and mechanical processes underlying the adverse effects of vibration must begin with an investigation of the mechanical properties of tissue. Hence, the literature was reviewed in the following areas:

- material properties of the spine;

- nutrition and fatigue failure of the spine;

- loss of stature - spinal creep; and

27

- impact acceleration and spinal injury.

The spine is a complex structure consisting of a series of rigid elements (vertebrae) connected by flexible visco-elastic units (intervertebral discs). Compressive, bending and shear loading are transmitted throughout the spine by a combination of forces in the intervertebral discs, apophyseal facet joints, ligamentous structures and active muscle contraction. Whether a person is standing, walking, or seated, the intervertebral disc is subject to stress.

Numerous hypotheses were reviewed with respect to the etiology of back disorders. One of the frequently cited hypotheses suggested that vibration alters nutrition of the disc (Dupuis and Zerlett, 1986). A second suggested that dynamic loading of the intervertebral joints causes fatigue damage to the annulus of the intervertebral discs (Sandover, 1981).

An important difference between a purely mechanical system and a living system is that the mechanical system does not change under constant stress - provided that the strain does not exceed the elastic limit. In a biological system, the elastic properties of tissue are a time-dependent function of the applied stress. Thus, loss of fluid takes place from the intervertebral disc space in response to static loading, resulting in a loss of stature (referred to as creep). This affects the stability of the spinal unit and causes a redistribution of stresses in the surrounding tissue. Unlike mechanical structures, the properties of biological materials are also a time dependent function of their nutritional status.

Investigations of spinal units in-vitro revealed non-linear load-deflection characteristics whereby the ultimate strength and stiffness increased with the rate of compression. Fracture of the end plate occurred within the elastic limit of the material. In general, failure occurred due to compressive fractures of the vertebra, while the intervertebral disc remained intact. In single impact studies, vertebral damage occurred most frequently in the lower thoracic and upper lumbar region in the form of anterior wedge fractures, at shock accelerations of 18 to 25 g. Chronic degenerative failure in response to WBV was attributed to both nutritional and fatigue mechanisms.

In the literature, calculation of fatigue failure properties of the spine were based on in-vitro data. They did not include consideration of the ability of tissue to recover or repair through on-going nutritional mechanisms. Hence, due to the absence of a regenerative model, these calculations may underestimate the real fatigue life of tissues in-vivo.

Although the literature appeared to report distinct differences in the mechanisms of acute and chronic injury, it is probable that both types of injury are a function of material behavior. Based on this conclusion the material properties of tissue, and both the mechanisms of acute impact injury and chronic degenerative failure were recommended for incorporation into a unified theory for mechanical injury leading to the HHA method in Phase 5.

Biodynamic models

Biodynamic models which were reviewed were classified according to either the purpose of the model, or the function to be modeled (von Gierke, 1971). The most common purposes identified were:

- to understand basic pathological processes, physiological responses, or biomechanical responses to various mechanical stresses;

- to predict human response to stress in circumstances where experimental data is either unavailable or unobtainable; and

- to determine the engineering design of systems to provide protection, comfort or safeguard performance of the operator.

In summarizing the status of biodynamic models, Griffin (1981) provided the following assessment:

- most models are based on inadequate experimental data;

- current models are highly restrictive in their application; and

- models devised for different purposes (for example spinal injury or performance) may have little in common.

Griffin (1981) also commented that the extent of human variability and shortage of experimental data make it possible for a one degree of freedom model (such as the DRI) to compete successfully with much more sophisticated 3-dimensional or discrete parameter models. Despite this criticism, several important contributions to biodynamic modeling have direct relevance to the development of a health hazard index, for example:

- Orne (1969) has shown that the introduction of anterior-posterior input forces, and the presence of bending moments (and hence flexion) within the spine, substantially alters the prediction of compressive stresses acting on the thoracic and lumbar motion segments.

- Prasad and King (1974) have shown that the articular facets play an important role in the transfer of compressive forces during axial impact loading.

- Hinz and Seidel (1989) have shown that any fatigue model based on rms values of acceleration, as an estimate of input stress, will underestimate the health effects of vibration. This is due to the non-linear nature of the transfer function between the input acceleration at the seat and the output acceleration wave form.

- Sandover (1983) has proposed a model based on fatigue failure of materials. Sandover selected data on the fatigue characteristics of bone and cartilage to model fatigue failure of tissue in response to cyclic loading. Models of both vertebral end-plate and the disc annulus suggested the possibility of fatigue failure in those structures.

- Seidel, Blüthner and Hinz (1986) constructed a model of stress in the lumbar spine based on anthropometric data, EMG activity and accelerations of the upper trunk (measured at the thoracic vertebra). The predictions of this model also supported the possibility of fatigue failure at the end plates of lumbar vertebrae after long term exposure to WBV.

Standards and guidelines for shock and vibration

To provide a proper historical perspective to the development of the HHA, the main standards and guidelines governing exposure to vibration and shock were reviewed. In the Phase 1 report, the evaluation of each standard or guideline contained a summary of the experimental and computational techniques, and where appropriate, a discussion of the limitations of the standard (Village et al., 1995a).

Included in the Phase 1 report (Village et al., 1995a) is a review of the following standards and guidelines:

- ISO 2631 (1974): Guide to the Evaluation of Human Exposure to Whole-Body Vibration;

- ISO 2631-(1978); Amendment 1 (1982) and Addendum 2 (1982);

- ISO 2631/1 (1985) and 2631/3 (1985);

- ISO 2631/2 (1989) Evaluation of Human Exposure to Whole-Body Vibration - Part 2: Continuous and Shock Induced Vibration in Buildings.;

- ISO/TC 108/SC4 (1991) Third Committee Draft Revision;

- British Standard 6841 Measurement and Evaluation of Human Exposure to Whole-Body Mechanical Vibration and Repeated Shock 1987;

- Verin Deutscher Ingenieure (VDI 2057, 1986);

- Dynamic Response Index (DRI) (Payne, 1965; revised 1992);

- Air Standards Coordinating Committee (ASCC 1982);

- Hazard Dose Value (Griffin, 1982);

- Japanese developments (Kanda et al., 1982);

- Other Methods Using Biodynamic Models;

- Janeway's Criteria;

- Absorbed Power; and

• USSR Methods.

The most recognized standard for human response to WBV is the International Standards Organization (ISO) 2631. However, lack of consensus among members of the technical committee (TC 108/SC4) impeded publication of a major revision of the ISO 2631 for several years. (Note that the ISO 2631 revision that was released in July, 1997, was not included in the review of literature). ISO Draft Revisions appeared similar in many respects to the British Standards (BS 6841, 1987). These new standards incorporated rmq and VDV as the main method of characterizing vibration with shocks. Limits of over-exposure were removed from the body of the standards and placed in Appendices as guidelines.

Both the ISO Draft Revision (1991) and the BS 6841 state that epidemiological evidence supports the 4 to 8 hour vertical acceleration limit of the previous ISO 2631 Standard (1982), and that this is roughly equivalent to a VDV of 15. However, little evidence exists to support use of these guidelines for signals with repeated shocks of high magnitude.

Two guidelines were found to rate the exposure to repeated shocks (Air Standardization Coordinating Committee, 1982; Kanda et al., 1982). Both use the dynamic response index (DRI), a model predicting spinal injury, as their basis. The Air Standardization Coordinating Committee curves of severe discomfort plot the number of shocks in 24 hours as a function of the DRI. A different curve plots the 5% injury probability over a 100 day recovery. Kanda et al.'s tentative daily exposure is based on a study of spinal disorders among crew members of high speed ships. It is not clear from the latter study how their limit curve was derived. Although validated with health data, both guidelines are limited to models of spinal injury and to repeated shock in the vertical axis. None of the standards consider recovery explicitly in their models.

Vibration data collected in the field

Vehicle data collected in field studies were reviewed in the following categories:

• wheeled vehicles;

• shock and vibration in off-road, heavy equipment;

32

- shock and vibration in air transport; and

- shock and vibration in miscellaneous environments.

Despite the efforts of the ISO to standardize measurement and analysis of WBV data, a wide range of methods exist to assess the motion characteristics of vehicles. Where possible in the review of literature, data from studies were converted to rms measures, and weighted according to the curves in ISO 2631 (1978). By standardizing these data, the motion of different vehicles could be compared directly.

Tables were constructed to compare various categories of:

- on-road wheeled vehicles (buses, cars and trucks);

- off-road heavy equipment (tractors, bulldozers, skidders and fighting vehicles);

- aircraft (helicopters and fixed wing); and

- miscellaneous vehicles (ships, motorcycles, subways and trains).

Some of the highest levels of vertical acceleration and crest-factors were reported for military vehicles. The large range in acceleration levels were a reflection of the following factors: different measurement procedures; types of terrain; speed of travel; experience of driver; instructions given to the driver; whether the equipment was loaded or unloaded; and the total number of measurements taken.

Signal processing

Advanced mathematical methods were required in this study to quantify the complex acceleration wave forms generated by vehicles and to allow their effect on the human body to be assessed. With respect to signal processing, the literature was examined in the following categories:

- data acquisition;

- frequency domain analysis and stationarity;

- other methods of spectral estimation;

- time-frequency representations;

33

- time domain analysis; and

- techniques for separating shocks from continuous, random vibration.

To better understand signal processing techniques applicable to this project, the literature review explored combinations of analytical techniques in the frequency domain and the time domain which were later used to construct a vibration dose.

Time domain techniques, combined with appropriate frequency weightings and temporal assessments of the vibration exposure, were identified as the most promising to characterize vibration signals containing mechanical shocks and repeated impacts. Conventional frequency domain analysis techniques were selected for validation and for providing a reference when comparing to existing data. These mathematical methods were utilized extensively in Phase 2 for the characterization of the motion environment of vehicles.

Phase 1 conclusions

The following conclusions were developed from the review of literature:

Acute and chronic health disorders

- Epidemiological studies of WBV have investigated both subjective symptoms and objective measurements of health disorders. Unfortunately, most studies are poorly designed. Thus, determining well-supported cause-effect relationships between exposure to WBV and health disorders is difficult.

- Evidence exists that long-term exposure to vibration can be harmful to the spine, and possibly gastrointestinal and cardiovascular systems. Rather than causing specific pathologies, vibration seems to accelerate the onset of currently recognizable syndromes.

- There appears to be distinct differences in the mechanisms of acute and chronic injury to the spine caused by exposure to shock and vibration. Severe mechanical shock causes fracture of the vertebrae, most commonly in the lower thoracic and upper lumbar region.

Chronic back problems are usually in the lumbar region. Vibration-induced nutritional and fatigue mechanisms have been hypothesized to explain the etiology of back disorders.

Vibration measurement and standards

- A large number of studies have involved subjective response to vibration. Much of the data have been used in formulating the ISO guidelines for human response to WBV.

- Levels of vibration and shock have been collected from a wide range of studies. Some of the highest levels of vertical acceleration and crest-factors were reported for military vehicles. When evaluated using the ISO 2631 (1982) guidelines, some of these vehicles exceeded the recommended exposure limits within minutes.

- Recent Draft Revisions of ISO 2631, and the British Standard (BS 6841, 1987) incorporate rmq and the VDV as the preferred method of characterizing vibration with high crest-factors. Little evidence exists, however, to support use of these standards for signals with repeated shocks of high magnitude. Exposure limits are not contained in the body of these standards.

- Two guidelines exist for exposure to repeated shocks, both using the DRI (a model predicting spinal injury) as their basis (Air Standardization Coordinating Committee, 1982; Kanda et al., 1982). The DRI is limited to shocks in the vertical axis, with no consideration of recovery. The predictive power of the DRI has been questioned, and there is no independent validation of Kanda et al.'s guideline.

- In the few investigations that involved subjective response to shock or repeated impact, the rms and rmq measurements seemed equally good predictors of discomfort; the exponent of best fit depends upon the frequency, separation and anticipation of the shocks, background vibration levels, and prior training.

- A number of signal processing techniques have been used in other applications which may prove useful in characterizing impulsive signals from TGVs.

35

These include shock spectrum, peak processing and
auto-regression techniques.

Human response to vibration and shocks

- Many of the physiological and biochemical responses to
  WBV mimic those produced by exercise, including general
  stress, muscle fatigue and tissue or organ damage.
  Few studies in humans have investigated these responses
  in conditions of repeated shocks, and none have
  investigated the body's ability to recover. Similarly,
  theories of fatigue failure do not consider tissue
  recovery through on-going nutritional mechanisms.

- Measurements of impedance and apparent mass provide
  useful indications of the response of the body to WBV and
  repeated shocks. However, these measurements do not
  provide sufficient detail to determine the behavior of,
  or stresses acting on, individual systems such as the
  abdomen and spine.

- Biodynamic models range from single degree of freedom
  to three-dimensional and discrete parameter models.
  Sophisticated models suffer from lack of experimental
  data to validate their predictions. Most models are
  not designed to predict chronic health problems.
  Despite these criticisms, certain biodynamic models
  have direct relevance to development of a HHA.

Phase 1 recommendations

Recommendations for the health hazard assessment methodology

- Chronic health effects from epidemiological literature
  suggest that the spine and gastrointestinal systems are
  most affected by WBV and repeated shocks. Measurements of
  stress in these systems during simulated TGV exposures
  can be compared with "safe" stress induced in more
  familiar environments (walking or light physical work) to
  assess potential for accelerated onset of chronic health
  problems.

- Physiological, biochemical and biomechanical data will be
  used to indicate deviation from normal responses, to seek

36

correlations with individual and repeated shocks, and to measure recovery following shocks.

- Physiological stress may be evaluated with electrocardiograph (ECG) spectral components, responsiveness of ECG parameters to repeated shocks and biochemical markers such as LDH and CPK activity. EMG measurements and biochemical markers such as serum electrolytes can indicate muscle fatigue. Tissue damage can be investigated by measurements such as von Willebrand's factor.

- Correlations between physiological, biochemical and biomechanical changes in response to shocks will be used to identify possible chronic health problems.

- Measurements of spinal acceleration, abdominal displacement and muscle tension (EMG) can be used in biomechanical modeling to calculate internal tissue stresses in response to shocks. It may be possible to devise a unified theory of injury which will encompass the mechanisms of both acute damage and chronic degenerative failure.

- Since available standards and guidelines do not accommodate the high crest-factors and acceleration levels measured in TGVs, there is an obvious requirement for signal characterizations capable of encompassing both impulsive accelerations and multi-axial random vibration.

- Time domain techniques, combined with appropriate frequency weightings and temporal assessments of the vibration exposure, appear to be the most promising means of characterizing signals containing shocks and repeated impacts. Methods which merit consideration include higher order means, dose measures, peak processing methods, auto-regression and cumulative damage models. Conventional frequency domain analyses are important for validation and for providing a reference when comparing to existing data.

- Although a subjective response model may be acceptable for exposures to random vibration, it becomes difficult to apply with confidence to shock and repeated impact. In these circumstances, a biodynamic model may provide a more versatile prediction of human response. Advances in

37

modeling offer the potential of an index incorporating
spinal loading, tissue stresses, and fatigue and recovery
characteristics of various systems of the body.

- Epidemiological data of health disorders, combined with
vibration levels of various equipment, may help to
construct dose-response relationships that can assist
in validating the eventual HHA method.

Measurement indices for pilot test by priority

From the review of literature, it was apparent that few
studies have exposed humans to vibration with repeated shocks in
a controlled laboratory environment. This type of experimental
control is essential to study the physiological, biochemical and
biomechanical responses of the body to motion environments and
to use these data to develop exposure guidelines.

Several measures were selected for inclusion in the pilot
study (Phase 3) in order to evaluate the human response to
vibration and repeated shock.

- Triaxial seatpan accelerations

- EMG of paraspinal muscles

- Biochemical markers: LDH, CPK activity, von Willebrand's
factor, hemotological factors, serum electrolyte

- ECG: spectral and temporal components

- Triaxial spinal accelerations

- Gastrointestinal pressure or displacement

- Shoulder/head acceleration

- Subjective assessment

Measurement of gastrointestinal motility, peripheral blood
flow, and temporary threshold shift were also considered,
but ultimately excluded from the pilot study.

Phase 2: Characterization of the environment

Phase 2 introduction

The objective of Phase 2 was to characterize the repeated shock environment of TGVs through analysis of recorded acceleration signatures, and to develop methods for realistic simulation of the TGV acceleration environment.

In order to accomplish the objective of Phase 2, the following tasks were completed:

- theoretical representation of human exposure to shock and vibration;

- analysis of vehicle seat shock and vibration data; and

- vibration and shock simulation.

The development of the theoretical framework with which to characterize vehicular motion was mathematical in nature. In the Phase 2 report, details are provided of the analytical methods for characterizing the vibration, shock and repeated impacts to which occupants of TGVs may be exposed (Roddan et al., 1995). As well, the rationale which lead to the choice of the equations presented in this document are examined.

Of primary concern to this study is the specification of human response to vibration containing mechanical shocks, jolts or impacts. In order to evaluate the human response, it was first necessary to characterize and simulate WBV and repeated shocks.

The term "shock" is used to describe a transient or impulsive mechanical input to the human body (force, displacement, velocity and/or acceleration) that causes forced disturbances in the relative positions of body parts. Such relative motion of body parts may result in excessive strains within tissues, ligaments or bones.

Vibration consists of oscillations that are characterized by zero mean displacement, or rotation. There is generally no net translational motion, though the oscillation may be referred to a moving frame of reference (e.g., a moving vehicle). The extent

39

of the oscillation defines the amplitude of the motion, while the (time) rate of oscillation defines its frequency.

For convenience and clarity, several types of oscillatory motion are commonly distinguished in the literature. When the character of future oscillations may be determined by past oscillations of a system, the motion is considered to be deterministic in nature as illustrated by the "sinusoidal" or "multi-sinusoidal" waveforms in Figure 1. Deterministic waveforms, or signals as they are commonly called when discussing aspects of signal processing, may occur as periodic (e.g., sinusoidal) or non-periodic motion (e.g., the transient and shock waveforms in Figure 1).

A second class of signals (considered non-deterministic) are those in which future motion of a system is unrelated to its past motion. This class of signals includes random vibration. The properties of random signals must be described statistically. The basic characteristics of such signals are described as: stationary (or ergodic), that is, with amplitude properties that are statistically time independent (e.g., the stationary random signal in Figure 1); or non-stationary, that is, with time-dependent waveform statistics, as displayed by the non-stationary random signal in Figure 1.

The classification of physical data as being either deterministic or random may be argued in some cases. The signals shown in Figure 1 were representative of the forms of seat motion observed, and provided a framework for discussion of the signal processing methods developed in Phase 2. In reality, combinations of these signals frequently occur. This was considered in the establishment and selection of appropriate acceleration amplitudes and frequencies which were necessary to determine human responses and health effects.

Theoretical representation of human exposure to shock and vibration

Vehicle motion over uneven terrain results in seat motion (displacement and acceleration) that contains random and deterministic components. When the latter contain impulses or shocks, methods of analysis for the assessment of human responses, including health effects, commonly differ for these two components.

Several methods for characterizing individual and repeated shocks, with or without background vibration, were described which involved both statistical and waveform based measures. The latter measure provided a model suitable for shock waveform synthesis. These were used first to characterize the seat motion in TGVs, and then to simulate common features of this motion.

Magnitude characterization

The literature usually expresses WBV as the root mean square (rms) value of acceleration in a specified time interval, and peak vibration is described as a crest factor. Methods to quantify the magnitude of background vibration and shocks or impacts were described, and a simple model was developed to describe shock waveforms that could be used in simulations.

Frequency characterization

Because the human response to vibration depends on the frequency of the motion, a method to determine the frequency characteristics was described. This allowed a motion signal to be described in terms of a frequency weighted acceleration, which best describes its vibration hazard. This was also important in order to compare motion signatures to standards, guidelines and biodynamic models which predict the response of the human to WBV or shocks, such as:

- frequency weightings specified by International Standard ISO 2631 (1978) and 2631/1 (1985);

- frequency weightings specified by British Standard BS 6841 (1987);

- the Fairley-Griffen model (1989) which is representative of the human response to small amplitude vibration; and

- the Payne model which was developed to assess human exposure to shocks. The Payne model is usually described in terms of the DRI which has widespread acceptance for evaluating spinal injury from exposure to large amplitude single shocks in the z direction (Payne, 1975).

Integrated dose measures

The purpose of an integrated dose measure is to provide, in a single formula or procedure, a unified treatment of vibration exposures. The method is independent of the nature

41

of the motion experienced by the body - be it continuous, intermittent, random, periodic, transient, single or multiple shocks, and single or multi-axis. While the specification of such a measure may appear to be an ambitious goal, it has been attempted in some standards (e.g., BS 6841, 1987), and in the derivation of some dose measures (e.g., Griffin, 1982). Although there is no evidence in the literature to determine whether the same measure will apply to different health effects, and/or subjective responses to vibration, the advantages of an integrated dose measure are self-evident.

The concept of an integrated dose measure provided a framework for assessing the exposures recorded in TGVs, and for interpreting the laboratory experiments. A generalized dose function (D) was developed for exposure to shocks and/or vibration for an extended period of time (T) (which may encompass a workday) from a single-number measure of the magnitude of the hazard at time (t), for example the frequency weighted acceleration $a_w(t)$.

$$D(a_w,T)_{m,r} = \left\{ \int_0^T [a_w(t)]^m dt \right\}^{\frac{1}{r}}$$

(1)

In this expression, m and r are respective constants representing the moment and root of the dose measure.

In order to establish an estimate of the dose using this method, it was necessary to determine an appropriate measure of the following:

- the magnitude of the stimulus;

- the relative hazard presented by vibration at different frequencies; and

- the combined effect of vibration in different directions.

After establishing the magnitudes of vibrations at different frequencies that are considered to be equally harmful (by means of frequency weighting functions), dose models were described that could be employed to quantify the health effects from exposure to repeated shocks and vibration. The potential for biological recovery processes (i.e.,time dependent tissue properties such as visco-elasticity or repair mechanisms) to

42

occur both during and after exposure were also introduced into the model.

The most recognized dose measures that may be employed to quantify the health effects from exposure to repeated shocks and vibration were described in detail in the Phase 2 report and are summarized in the following subsections (Roddan et al., 1995).

Dose measures in which moment "m" is equal to root "r"

For translational motion containing both shocks and vibration, the dose measure with m=r=4 has been proposed by Griffin as the preferred measure of human response Griffin, 1984). In Griffin's terminology, the vibration dose value (VDV) is given by:

$$VDV = D(a_W, T)_{4,4} = a_{W(rmq)}[T]^{\frac{1}{4}}$$ (2)

This relationship, and successively higher-order dose functions, are related to increasingly higher-order root mean values. The latter more closely approximate the peak value of a waveform. Hence the higher-order dose functions give progressively more emphasis to shocks in comparison with random vibration. This property of higher-order dose functions was directly relevant to the objectives of the present study, and so dose functions with moment and order of up to 12 were employed in the analysis of seat motion data.

Dose measures in which moment "m" is not equal to root "r"

Integrated dose functions for exposure to shocks and vibration may be constructed in which m≠r. Dose functions in which m>2 and r=1 have been explored by Griffin (1982) and Wickström, Kjellberg, and Dallner, (1990). In general, the properties of these functions are equivalent to those of the preceding section with the same value of m. However, relationships between dose functions with different values of m are significantly affected. For example, the relationship between the two lowest-order dose functions becomes:

$$\frac{D(a_W, T)_{4,1}}{D(a_W, T)_{2,1}} = \frac{a_{W(rmq)}^4}{a_{W(rms)}^2}$$ (3)

For Gaussian random vibration, this equation becomes, by virtue of the numerical relationship between rmq and rms:

$$\frac{D(a_w, T)_{4,1}}{D(a_w, T)_{2,1}} = 3a_{W(rms)}^2 \qquad (4)$$

A dose measure for head injury from impacts has been proposed in which m=2.5 and r=1(Griffin, 1990). This measure, which employs the acceleration time history of the impact without applying any frequency weighting, has been developed into a severity index that has been incorporated into regulations for vehicle equipment and helmets (Versace, 1971).

The development of dose measures that include recovery mechanisms was of considerable interest, for inclusion in the dose-effect models in Phase 5. The dose measures introduced so far, while integrating the effects of exposure to oscillatory motions with different magnitudes and waveforms, do not account for the temporal pattern of hazardous events. Thus, such dose measures do not allow for the existence of biological recovery mechanisms that may mitigate the potential health effect some time after an event has occurred, or while exposure to WBV and/or shock is continuing.

The generalized dose function of equation 1 was modified to account for the sequence of individual hazardous events, by segmenting the acceleration time history from which it was constructed into a time series. The dose elements were listed in the reverse order in which they were experienced. Hence, the health effect resulting from individual dose elements could be weighted to allow for recovery processes (Chatfield, 1989).

Dose estimates for exposure to shocks

The development of dose measures for human exposure to shocks appears to have proceeded independently of comparable measures for WBV. The former focused on the time history of the shocks, in order to quantify specific health or injury issues (e.g., head injury during vehicle crash, or back injury during pilot ejection). The latter tended to focus on responses to different vibration frequencies in order to quantify issues such as discomfort. In Phase 2, measures were introduced that have been used for the assessment of spinal injury in a seated person.

These measures considered single or repeated shocks from motion in the z axis. An integrated dose model was also described which would utilize such measures.

Exposure to single shocks

The potential for injury to the spine from rocket-propelled aircraft ejection seats led to the development of the DRI (Payne, 1975). The DRI is a single-degree-of-freedom biodynamic model of the human spine with supporting structures. The magnitude of the index, from which the potential for spinal injury is derived, is based on the maximum compression of the spring of the model in response to the shock waveform input to the base. The maximum compression of the spring corresponds to the peak strain on the spine in the z axis. This is expressed in the form of a non-dimensional index by:

$$DRI = \frac{\omega_n^2 \Delta_{max}}{g} \qquad (5)$$

where ω_n^2 is the natural frequency, Δ_{max} is the maximum spring compression, and g is the acceleration due to gravity.

An equivalent measure for evaluating exposure to the less intense shocks expected to occur during TGV operation may be constructed using a biodynamic model that better represents the response of the upper body to less intense seat motion. Such models have been derived from the measured dynamic response of the human body (Fairley and Griffin, 1989), or proposed for various body subsystems (Allen, 1978b). The Fairley-Griffin model was chosen in this study to provide an analysis of seat motion based on a biodynamic model.

Exposure to repeated shocks

The concept of the DRI was extended by Allen, and others, to the evaluation of repeated shocks experienced by seated persons in the z axis (Allen, 1977, 1978a, Payne, 1991). For this purpose, Allen (1997) proposed a relationship between the severity and number of repeated shocks. The severity was expressed by the magnitude of the DRI, while the number of shocks was measured over a 24 hour period. Equi-noxious contours were then constructed for the risk of spinal injury and discomfort for seated persons (Allen, 1977). A procedure for implementing this relationship was adopted by the Air Standardization Coordinating

45

Committee (ASCC), though the description is open to interpretation (Air Standardization Coordinating Committee, 1982).

Although the ASCC criterion for human tolerance to repeated shock is expressed in terms of the DRI magnitudes, the Palmgren-Miner cycle-ratio summation hypothesis, on which the ASCC state their procedure to be based, concerns the number of cycles, or in this case number of shocks (Shigley and Mitchell, 1983), and not the stress level. The hypothesis, which was developed to account for the fatigue life of metals, is commonly expressed as:

$$\sum_{q=1}^{Q} \left[\frac{n_q}{N_q} \right] \leq 1 , \tag{6}$$

where n_q is the observed number of impacts, and N_q is the maximum number of impacts to failure.

Implementation of this equation requires counting the number of shocks that fall within prescribed ranges of DRI values. The number is then compared to a predetermined maximum allowable number for each DRI range. A logarithmic relationship exists to relate the applied stress level and the number of cycles to failure of the form:

$$n_q = [\sigma_\mu / \sigma_q]^a \tag{7}$$

where σ_μ is the static failure stress and σ_q is the applied stress level.

Hence, if the Palmgren-Miner cycle-ratio summation hypothesis is employed to define the acceptability of exposures expressed in terms of the DRI, then the term in square brackets must be raised to the power a. Allen (1978) obtained a value of a=8 from some very limited air crew data and physiological data relating to the performance of NATO tank crews. Sandover (1986) subsequently pointed out that a=8 is also a good approximation for the fatigue of biological material (citing Weightman (1976 a, b) for cartilage; and Carter et al. (1981) and Lafferty and Raju (1979) for bone). The criterion of acceptability thus becomes:

$$\sum_{q=1}^{Q} \left[\frac{(DRI)_q}{(DRI_{max})_{n_q}} \right]^8 \leq 1 \tag{8}$$

46

Dose model with recovery processes

A conceptual framework was developed to construct a model for quantifying the health effects resulting from exposure to repeated shocks and vibration which included consideration of the following:

- the underlying physical property of oscillatory motion responsible for back injury or pain which appear to be related to the compressive, and/or bending strain induced in the spine;

- a one-dimensional dose model that could be based on a measure of strain derived from a one degree of freedom biodynamic model, where strain is related to the net compression of the spring of the model, and may be expressed in terms of the base and mass displacements;

- a dose function for exposure to repeated shocks, in which the tolerable spinal compression expressed by the magnitude of the DRI is related to the maximum daily number of impacts;

- studies of the fatigue failure of human bone with strain rates reversing approximately once per second; and

- biological recovery processes.

By employing a generalized dose function, the integrated effect of exposure to shocks of unequal magnitude and shocks irregularly spaced throughout the day can be calculated using a plausible relationship between shock magnitude and number. A dose function of this form may be formulated to represent exposure to random vibration, and therefore offered the potential for constructing an integrated dose model in Phase 5.

Analysis of vehicle seat impact and vibration data

In order to characterize and simulate the vibration environment experienced by military personnel, it was first necessary to obtain reliable seat motion data for a wide range of vehicles and operating conditions. A primary requirement was that the data not only possess a range of continuous (background) vibration signatures, but that they also contain a sufficient number of impact signatures in all three axis. Statistics

47

derived from these data were then used to construct realistic experimental test signals for use on the MARS.

Data were supplied from two main sources. The first source was the Waterways Experimental Station (W.E.S.) laboratory located near Vicksburg, MS. The vibration data obtained from this laboratory were recorded in the z axis only, and therefore provided an incomplete record of typical operating environments. Because of this limitation, additional data that contained simultaneous vibration information in the x, y and z axes from a range of TGVs were obtained from US Army Aberdeen Proving Ground in Maryland.

The vehicles tested included the FAV, the M2 Bradley, the M1A1, the M1A1 HTT, the M1026 HMMWV, the M109A3, the M923A2, the XM1076, and the M2HS Bradley. All seat motion data had been recorded by seat-pad accelerometers mounted in a seatpad between the buttocks and the seat as specified by ISO 2631 (1985) and BS 6841 (1987).

The shock and vibration data were analyzed. A complete listing of the programs that were written to process the data are described in the Phase 2 report (Roddan, et al., 1995).

The seat motions which were recorded in a range of TGVs during off- and on-the-road operations were described, together with values of key measures derived from these exposures. A procedure involving computer recognition of impulses, including shocks and other transient or non-stationary motions, within a background of stationary (Gaussian) random, or near-sinusoidal, vibration was used to classify vehicle seat motion into the following four categories:

Type 1   Gaussian random motion (Figure 2);

Type 2   Periodic deterministic motion, which is typically dominated by tonal (narrow-band) components and may be amplitude modulated (Figure 3);

Type 3   Intermittent motion - non-stationary random and transient deterministic signals (Figure 4); and

Type 4   Impulsive motion including shocks (Figure 5).

The ranges of mean rms acceleration, frequency weighted according to BS 6841 (1987), observed for type 1 and type 2 seat motions are listed in Table 5.

Table 5
Range of frequency weighted rms acceleration for Type 1 and Type 2 seat motions

Axis	Type 1	Type 2
x	0.08 to 0.69 $m \cdot s^{-2}$	0.11 to 2.01 $m \cdot s^{-2}$
y	0.09 to 0.57 $m \cdot s^{-2}$	0.08 to 0.60 $m \cdot s^{-2}$
z	0.11 to 2.53 $m \cdot s^{-2}$	0.15 to 3.14 $m \cdot s^{-2}$

Summaries of all seat-motion data, frequency weighted according to the British Standard 6841, the ISO Standard 2631, and the Fairley-Griffin Model, are tabulated as Appendices to the Phase 2 report (Roddan et al., 1995). The seat motion data recorded during cross-country operations are also summarized separately.

Vibration and impact simulation

A method was developed to synthesize the shock and background vibration typical of seat motion in TGVs for use in Phase 3. A number of different shock signatures were constructed in which the amplitude, rise time (frequency), number of shocks per minute, and background level of vibration were varied. Shock amplitudes ranged from 0.5 to 3.0 g, with a fundamental frequency of 2 to 11 Hz. Shock rate ranged from 1 shock per 2.5 minutes (at 3 g) to 32 shocks per minute (at 1 g and 2 g). The signatures developed for Phase 3 ranged in duration from 5.5 minutes to 2 hours. The signatures were intended to be representative of seat motion observed in the military vehicles.

Phase 2 conclusions

A conceptual framework was developed with which to quantify the salient parameters of seat motion in TGVs, and to integrate these parameters into measures of shock and vibration exposure. The essential elements of a dose model for quantifying health effects resulting from exposure to repeated shocks and vibration, including the potential for biological recovery processes, were identified in a form suitable for machine computation.

A procedure involving computer recognition of impulses, including shocks and other transient or non-stationary motions, within a background of Gaussian random, or near-sinusoidal vibration was developed and used to classify TGV seat motion into four the following four types:

Type 1   Gaussian random motion;

Type 2   periodic deterministic motion;

Type 3   intermittent motion; and

Type 4   impulsive motion, including shocks.

A classification of shock waveforms by initial peak amplitude, fundamental frequency, and decay rate was developed and used to analyze the shocks recorded at the seats of TGVs, from which representative shocks could be deduced. In this classification, a type +1 shock consists of a single positive motion, with amplitude greater than 1 g, followed by a single negative oscillation; a type +2 shock consists of an initial positive motion, with amplitude greater than 1 g, followed by oscillatory motion that included a second positive oscillation.

The most frequently observed shock in the z axis during cross-country operation of TGVs was a type +2 shock. Shock types +1, -1 and -2 were also common. The fundamental frequency of shock waveforms ranged from 0.8 to 21 Hz. A minimum of three, and a maximum of eight such shocks per minute were observed. The maximum (peak) positive acceleration observed was 6.5 g, while the maximum (peak) negative acceleration
was −3 g.

The most frequently observed shock in the x axis during cross-country operation of TGVs was a type +1 shock. The fundamental frequency of the shock waveform ranged from 1 to 60 Hz. There were a minimum of 20, and a maximum of 60 of these

50

shocks per minute. The maximum (peak) positive acceleration observed was 3.7 g, and maximum (peak) negative acceleration was -3 g.

The most frequently observed shock in the y axis during cross-country operation of TGVs was also a type +1 shock, although +2 and +3 were also common. The fundamental frequency of the shock waveform ranged from 0.8 to 40 Hz. There were a minimum of 11, and a maximum of 29 such shocks per minute. The maximum (peak) positive acceleration observed was 4.0 g, and maximum (peak) negative acceleration was -1.8 g.

The shock and vibration environment at the seats of TGVs was simulated by combining two signals: one to characterize the shocks, and the other to characterize the near-continuous background vibration. The former was synthesized by a type +1 or type -1 shock, consisting of a damped sinusoidal waveform (Cameron et al., 1996: Phase 4 report, Figure 2) and the latter by a pseudo-random time series with a Gaussian amplitude probability density distribution.

Exposure signatures were created from the two types of signals and successfully run on the MARS facility for use in the pilot laboratory experiments. It was found that the idealized shock generated by computer tended to be modified by the dynamic characteristics of the simulator. The resultant shock waveform output by the table typically contained some superimposed high frequency noise, and a secondary damped oscillation preceding and following the main shock (see Figures 8 and 11). Shock and vibration signatures were constructed in which the amplitude, rise time (frequency), number of shocks per minute, and background level were varied. For Phase 3 experiments, shock amplitude ranged from 0.5 to 3.0 g, with a fundamental frequency of 2 to 11 Hz. Shock rate ranged from 1 shock per 2.5 minutes (at 3 g) to 32 shocks per minute (at 1 g to 2 g). The signatures developed for Phase 3 ranged in duration from 5.5 minutes to 2 hours.

Phase 3: Pilot studies

Phase 3 introduction

Potential indices for the prediction of exposure severity (and hence injury risk) were identified in the review of literature (Phase 1). Measures identified in Phase 1 were evaluated for inclusion in Phase 3 experiments. At this time, some were rejected from the protocol (i.e., gastrointestinal motility, peripheral blood flow and temporary threshold shift).

As part of Phase 3, pre-pilot studies were conducted at B.C. Research to develop and refine experimental techniques and standard operating procedures (SOPs) for instrumentation, data acquisition and equipment calibration. In the pre-pilot project, a single axis vibration table which provided controlled sinusoidal motion in the z axis was coupled with an impact device that delivered ± 1 g shocks in the z axis.

Phase 3 experiments, which were conducted in February and March 1993 at USAARL, Fort Rucker, Alabama, included short and long term exposure to motion signatures. The short term experiments consisted of a series of 5.5 minute exposures to differentiate the human response to individual shocks, while the long term experiments consisted of 1 and 2 hour exposures to background vibration with repeated shocks which were superimposed in the motion signatures.

The purpose of the pilot study was to determine the best experimental design to be used in Phase 4, and to provide quantitative information on the subject's tolerance to repeated shocks. This included appropriate motion signatures (in terms of the magnitude of shocks and exposure duration) and the selection of experimental measures that could provide the best human response data to correlate with the motion environment.

Phase 3 objectives and methods

The overall objectives of the pilot phase were:

- to select and test equipment and measurements to evaluate the biomechanical, physiological and biochemical responses to repeated shocks;

- to determine which measures of human response were most appropriate to include in Phase 4 experiments; and

- to establish appropriate motion signatures and duration of exposure for Phase 4.

Objectives of short term experiments

The objective of the short-term exposures was to evaluate the human response to individual shocks, including:

- a range of shock frequencies (from 2 to 11 Hz);

- a range of peak acceleration amplitudes (from 0.5 to 3 g);

- three biodynamic axes (+x, +y and -z);

- a variety in the number of shocks per unit time (2, 10 and 30 shocks per minute at 6 Hz);

- background rms vibration (0.05 to 0.4 g rms at 6 Hz);

- combinations of shocks and rms background vibration; and

- swept sinusoidal motion (0.3 g magnitude at 2 to 17 Hz)

Objectives of long term experiments

The objectives of the long duration exposures were:

- to examine time-dependent processes, including potential fatigue, injury and stress effects and recovery from repeated shocks;

- to compare the human response to exposures of different severity based on the vibration dose value (VDV); and

- to investigate whether the human response changes during prolonged exposure.

Methods

Following subject briefing, medical screening, and orientation, ten male subjects (aged 20 to 40) participated in the short term experiments. Table 6 summarizes the motion signatures used for short term exposures on the MARS. These experiments were designed to differentiate the effects from the following variables: shock magnitude; shock axis; rise time or waveform frequency; number of shocks per minute (shock number); effect of background rms vibration; and exposure to a single amplitude swept sine waveform.

Table 6
Design of short-term exposures

Experiment Number and Condition	Impact Number shocks·min^{-1}	Background rms Level	Shock Frequency (Hz)	Shock Amplitude (g)	Duration
Experiment 1 to 4 Rise Time	3	0.05 g broad band	2, 4, 6, 8, 11	0.5 g, 1 g 2 g, 3 g	5.5 minutes
Experiment 5 Shock Number	2, 10 or 30	0.05 g broad band	6 Hz	2 g	5.5 minutes
Experiment 6 Relationship of background rms versus shocks	2	0.1 g	6 Hz	2 g	5.5 minutes
		0.17 g	6 Hz	2 g	
		0.25 g	6 Hz	2 g	
		0.4 g	6 Hz	2 g	
Experiment 7 Response to swept sine wave	na	0.3 g	sinusoidal at 2 Hz to 17 Hz at 1/4 Hz per second	na	79 seconds

na= not applicable

54

To investigate whether the human response changes during prolonged exposure to motion signatures which include repeated shocks, four of the subjects were also exposed to long duration (1 and 2 hour) exposures with repeated shocks of 0.5 to 3 g having a waveform frequency of 6 Hz. Seven 2 hour exposures and two 1 hour exposures (with x and y axis shocks respectively) were presented to each subject. Details of each condition are presented in Table 7.

Table 7
Design of long-term exposures

Condition	Duration (hours)	rms (g)	Shock Amplitude & Direction	Shock Number
1. Control	2	none	none	none
2. Background rms	2	0.05x,y 0.16 z	none	none
3. Shocks + rms	2	0.05	1 g z axis	32 min^{-1}
4. Shocks + rms	2	0.05	2 g z axis	2 min^{-1}
5. Shocks + rms	2	0.05	3 g z axis	0.4 min^{-1}
6. Shocks + rms	2	0.05	2 g z axis	32 min^{-1}
7. Shocks + rms	2	0.05	1 g x, y, and z axes	32 min^{-1} random
8. Shocks + rms	1	0.05	1 g y axis	32 min^{-1}
9. Shocks + rms	1	0.05	1 g x axis	32 min^{-1}

Dependent variables

The dependent variables which were measured at regular intervals throughout the pilot experiments are described in Table 8. A detailed description of the techniques and equipment is provided in the Phase 3 report (Village et al., 1995b). Table 9 summarizes the variables included in each of the Phase 3 protocols.

Table 8
Dependent variables

Electrocardiography (ECG)	Electrocardiography involved taking a 12-lead ECG prior to and following vibration exposure, and a single lead ECG (3 electrodes) during vibration exposure.
Electromyography (EMG)	EMG was performed using disposable Ag/AgCl surface ECG electrodes placed bilaterally over the erector spinae muscles at L3 and T9. Abdominal EMG was measured from the rectus abdominus. EMG calibration and muscle fatigue procedures were performed using static back extensions against a force transducer.
Spine Acceleration	Acceleration was measured at the skin surface over the spinous processes of the lumbar and thoracic spine: x axis acceleration was measured at T1 and L2; y axis acceleration was measured at T2 and L3; and z axis acceleration was measured at T3 and L4.
Seatpad Acceleration	Triaxial acceleration (x, y and z axes) was measured at the seat by three accelerometers fitted into a seatpad that was positioned on the seat between the subject and the seatpan.
Internal Pressure	Internal pressure was monitored using a rectal probe containing a miniature pressure transducer.

Table 8 cont'd
Dependent variables

Abdominal and Thoracic Displacement	Abdominal and thoracic circumferential displacement was monitored during vibration exposure using an inductive coil sewn into belts worn at the chest and abdomen (Respitrace).
Positional Data from the Spine	Positional (Optotrak) data were collected using five infrared emitting diodes (IREDs) taped to the skin over the vertebral processes at C7, T4, T8, L1 and L5. A sixth marker was placed on the seat cushion.
Biological Specimens	Blood, urine and fecal samples were collected and analyzed in four of the two hour exposure conditions.
Cognitive Performance	A 15 minute cognitive performance test battery (Synthetic Work Environment) was performed during the long-term (1 and 2-hour) experiments. The battery consists of four tasks (Sternberg memory; stability tracking; 4-column addition; and auditory monitoring)
Subjective Response	Subjective response ratings were elicited for perceived discomfort, vibration, shocks, tiredness, and interference with task performance.

Table 9
Summary of dependent variables for Phase 3 experiments

Dependent Variable	Short Term	1-hour	2-hour
ECG	Y	Y	Y
EMG	Y	Y	Y
Spine Acceleration	Y	Y	Y
Seatpad Acceleration	Y	Y	Y
Internal Pressure	Y	Y	Y
Abdominal & thoracic displacement	Y	Y	Y
Optotrak	Y	Y	Y
Biochemical measures in biological specimens	N	N	Y
Synthetic work environment	N	Y	Y
Subjective response	Y	Y	Y

Y=Yes, N=No

Biological specimens (blood and urine) were collected and analyzed in four of the two hour experiments for evidence of the following:

- muscle damage;
- fluid shift;
- blood clotting;
- glucose in blood and urine;
- fatigue;
- inflammation;
- bone stress and remodeling; and
- kidney, bladder, or urinary tract dysfunction.

Phase 3 results and discussion

High frequency responses to individual shocks

Although the shock waveforms in the control signal for the MARS consisted of a damped sinusoid of a single frequency, the resultant output measured at the seatpad contained higher frequency components. This was most likely attributable to the resonances of the seat-subject sub-system mounted on the MARS. A sample of the unfiltered acceleration data measured at the seat and the spine in response to an x axis shock, is shown in Figure 6. Resonance was greatest in response to 4 Hz shocks at 3 g in the x and y directions, and decreased in severity with increasing waveform frequency and decreasing shock amplitude.

Resonance frequencies greater than 100 Hz observed at the seat were removed by low pass filtering the data. With a few exceptions, the high frequencies were not seen in the spinal acceleration records (Figure 7). However, the filtered seat acceleration record still contained significant frequency components above the nominal shock frequency of the control signal, which distorted the output waveform (Figure 8). By filtering at lower frequency cut-offs, the magnitude of the output was progressively attenuated. Generally, the differences were not large, and the relationship of response ratio to shock frequency at each shock amplitude remained essentially unchanged.

Vertebra-skin transfer function

Prior to analysis, the acceleration data were corrected for movement of the skin surface relative to the underlying bone (spinous process). This correction required a knowledge of the "bone-to-skin transfer function", which was derived for each subject for the y and z axis spinal accelerometers. A skin transfer function was not computed for the x axis accelerometers because they measured motion perpendicular to the skin surface and were not sensitive to shearing motion between the spinous process and the skin.

The bone-to-skin transfer function of each accelerometer was calculated as described by Hinz et al. (1988). Measured acceleration signals were then multiplied by their respective inverse transfer functions which were specific for each subject. This correction eliminates any contribution of bone-to-skin movement, and provides the true acceleration at the spinous process.

The calculation of the skin transfer function for the accelerometer-tissue sub-system was difficult. There was often an initial, very high frequency response on release of the skin, followed by lower frequency oscillations. A comparison of spinal acceleration measured at the lumbar level is shown in Figure 9 where the data are unfiltered, filtered at 60 Hz, and subsequently corrected by application of the inverse transfer function

The results suggested that the assumption of a simple Kelvin element for the accelerometer-tissue sub-system may be inappropriate for the analysis of spinal acceleration in response to shocks. Hinz et al. (1988) showed that the skin displays non-linear properties, and that it behaves differently at high frequencies. Evidence of this was found in the acceleration data obtained when the skin was pulled and released.

In some subjects, there was also evidence of high frequency vibration at the accelerometer when they struck the seat following a negative z axis shock. These oscillations may not have been transmitted from the vertebra to the skin surface. Instead, they may have resulted from a secondary resonance at the skin surface. Without filtering of the data to remove high frequency resonances, the inverse transfer function derived from a simple Kelvin element would severely distort the acceleration data calculated at the vertebra.

From the limited information available in this study, it was concluded that without a more complex model, the correction of acceleration data for skin movement probably does not improve accuracy, particularly for shocks where the subject leaves and then strikes the seat. Thus, a requirement was identified for more extensive skin transfer function analysis for Phase 4.

Spine acceleration, internal pressure and Respitrace responses

Spine acceleration

Despite the range of responses among individuals, the pattern of the mean response of spinal acceleration, internal pressure and Respitrace was remarkably similar. There were, however, differences in magnitude and shape of acceleration responses among the axes (x, y and z) and, in some cases, among the shock amplitudes. The largest spinal accelerations were observed in response to negative z axis shocks. Responses in the negative z axis were not, however, due to the initial shock

60

input, but rather to the subject impacting the seat. The acceleration response to positive y axis shocks was higher in magnitude than the response to positive x axis shocks. The data also showed a greater spinal response to 2 and 4 Hz shocks compared with 11 Hz shocks, as shown in Figure 10 and Figure 11.

The acceleration response in the positive x and negative z axes was non-linear with respect to the magnitude of the shock. Figure 12 illustrates the transmission ratio (i.e., the ratio of the spinal acceleration to the seat input) for the thoracic response to a negative z axis shock input. A non-linear response is apparent, with higher transmission ratios recorded for higher amplitude shocks. In response to y axis shocks, the spinal transmission ratio response curves were similar for all amplitudes at both lumbar and thoracic locations. The non-linearity observed for x axis shocks was not apparent in the y axis.

When the subject impacts the seat following a negative z axis shock, the seat accelerometer registers a very high-frequency component (approximately 150 Hz) (Figure 13). A high-frequency response (30 to 90 Hz) is transmitted to the accelerometers at the spinal vertebrae (Figure 14). Data in the literature suggests that the body damps these high frequency components (Fairley and Griffin, 1989). Our results showed higher spinal transmission of 2 and 4 Hz, compared to 11 Hz shocks. However, the much higher frequencies of 30 to 90 Hz were not being damped by the spine. These high frequency components might be interpreted as skin movement. However, the vertebra-skin transfer function did not remove them. In addition, examination of the internal pressure responses also showed a similar (i.e., high frequency) effect. It may be that for a large enough shock, the body responds as a non-linear system with high frequency components being transmitted through the body.

Internal pressure

Results from measures of internal pressure in response to various frequencies, amplitudes and directions of shocks are remarkably similar to spinal acceleration responses. Highest internal pressure responses were measured in the z axis (Figure 15). The internal pressure response produced by some 3 g shocks exceeded pressures that could be produced by subjects through forceful instantaneous effort (i.e., Valsalva maneuver).

Respitrace

Peak abdominal and chest displacement, which occurred in conjunction with shocks at the seat, were as high as 30 mm for individual subjects (Figure 16). Typically, displacement of the abdomen was greater than the chest. In many cases, displacement at the chest was negligible. For some subjects, especially in the z axis, no peak displacement was detected

Abdominal and chest displacement was calculated as a ratio of displacement (in mm) to seat acceleration (in $m \cdot s^{-2}$). In the case of abdominal displacement, the response ratios for 1 g shocks were generally higher than those for higher magnitude shocks. Thus, the pattern of abdominal displacement response to the magnitude of shock is opposite than found for other measures.

Assessment of ECG parameters

Vibration and shocks have been shown to affect the ECG signal. Changes have been reported in the R-R interval (Ullsperger, Seidel, and Menzel, 1986; Harada, Kondo and Kinura, 1990), heart rate variability (Harstela and Pilirainen, 1985), P-R interval (Abu-Lisan, 1979) and T-wave amplitude (Roman et al., 1968).

In this phase, analysis of the ECG was aimed at answering the following two questions: 1) what instantaneous effect do shocks have on the ECG R-R interval; and 2) does the long-term exposure to repeated shocks cause fatigue?

No strong fatigue effects were apparent during shock exposure or when compared to the control condition. There was no consistent difference between resting and recovery T-wave amplitude in any condition. Similarly, there was no discernible, consistent trend in R-R interval between or within conditions.

The effect of undertaking the performance task on spectral analysis of the R-R interval produced a decrease in variance. This effect is associated with increased effort and psychological stress. It is expected that undertaking cognitive tasks will affect the physiological response which may compound any effect induced by the shocks.

Heart rate differences were observed between experimental conditions. However, no strong trends were apparent over time in response to shocks. Because heart rate is affected by many different influences, including the psychological and physiological state of the subject, it was not considered a useful index for investigating fatigue due to repeated shocks.

Electromyography

Muscle response to individual shocks

The pattern of muscle response, observed as bursts and silent periods in the EMG, revealed the strategy used to compensate for the imposed motion of a shock. The main purpose of the muscle response to a shock acceleration is stabilization of the spine and upper body to minimize motion and preserve the seated posture. This maintains the individual's ability to continue performing the task at hand, and minimizes the effects of secondary impacts (through loss of balance or striking surfaces) that may prove harmful.

Evidence of two phases in the muscle response was seen in the EMG response to y and z axis shocks. The first phase is one of stabilization in which there is co-contraction of antagonistic muscle groups (both right and left erector spinae for y axis shocks and erector spinae and rectus abdominus for z axis shocks). The second phase is one of positional correction in which there is reciprocal activity of antagonistic muscle groups to correct for the imposed motion of the upper trunk and to restore neutral posture.

To determine if there was a difference either in burst onset, burst duration, or the pattern of bursting versus silent periods, the lumbar EMG burst responses to both positive and negative 1 g 6 Hz shocks in the x, y and z axes were examined. The direction of the shock influenced the resultant pattern of silent periods and bursts. A pattern of a burst followed by a silent period (shock-burst-silent-burst-silent) was observed for positive x axis and for negative z axis shocks. The opposite pattern of a silent period preceding a burst (shock-silent-burst-silent-burst) was observed for negative x axis and positive z axis shocks. In the y axis, the pattern of muscle response was dependent on the relation between shock direction and the side of the body from which EMG was monitored.

Figure 17 illustrates a typical lumbar EMG response to a negative 1 g, 6 Hz shock in the z axis. Burst durations ranged from 40 msec to 220 msec, however they were more typically in the range of 90 to 130 msec. No obvious differences were observed in the duration of the EMG bursts or silent period in response to shocks in various axes or directions. When multiple bursts were evident, the second burst tended to have a similar magnitude but longer duration than the first burst. The duration of silent periods ranged from 50 msec to 250 msec.

The magnitude of muscle response was dependent on shock frequency for each axis (Figure 18). However, each response was typically less than 10 percent of maximal voluntary contraction (MVC). These data support the pattern of frequency-dependence found in shock transmission data.

EMG-force calibration

A recommedation was made to improve the force calibration procedure that was employed in the Phase 3 experiments by eliminating the measurement of resting EMG and by increasing the number of measurements in the range of 2 to 10 percent MVC. In most cases, resting EMG data were larger than that measured during a 5 percent MVC. Because this was most likely due to the method of defining rest as zero force measured at the load cell. Because this condition may be achieved by leaning forward slightly, the muscle force required to support the mass of the upper torso is increased.

Fatigue

The mean power frequency (MF) and integrated EMG (IEMG) parameters measured during sustained isometric contractions before and after the long term exposures showed no consistent changes indicative of localized muscle fatigue. Failure to produce measurable muscle fatigue during test contractions may be due to the complexity of the musculature of the back, inadequate control over subjects' posture and muscle temperature, or to the selection of a sustained force that failed to produce significant fatigue within 30 seconds. Another consideration is that the muscle response to shocks was typically less than ten percent of MVC and lasted less than 200 msec. At this level of intermittent dynamic contraction, an appreciable amount of localized muscle fatigue would not be expected unless exposure to the motion environment was continued for a prolonged time period.

After a 2 hour exposure to 2 g, 6Hz z axis shocks at 32 shocks per minute, an increase in the magnitude of the lumbar EMG response was observed. This is consistent with an increased recruitment of motor units to produce equivalent force in a fatigued muscle. It is also consistent with observations of increased stretch reflex gain which results in a larger amplitude response to muscle stretch during fatiguing contractions (Darling and Hayes, 1983). An altered gain in the stretch reflex could be interpreted as a compensatory mechanism that maintains an appropriate level of force production despite muscle fatigue.

Optotrak displacement and acceleration

Analysis of the Optotrak data indicated that the system is capable of providing reliable acceleration responses to shocks of 1 to 3 g magnitude and 2 to 11 Hz frequency in the x and y axes. Although the system also measured the response to similar shocks in the z axis with reasonable accuracy, the sampling rates used did not capture the high frequency components which were superimposed upon the underlying shock waveform of acceleration measured by the accelerometers (Figure 19). These high frequency components only occurred in response to higher amplitude, low frequency shocks and were associated with the impact as the subject struck the seat rather than from the input shock waveform.

Preliminary trials with the Optotrak indicated that it was possible to collect three dimensional data from 6 markers at a sampling rate of 80 Hz. Subsequent analysis of data revealed occasional missing data (drop outs) in Optotrak data files. These missing data tended to coincide with the impact at the seat. Sample acceleration data comparing the output of an accelerometer and data derived from Optotrak output are presented in Figure 20.

The presence of missing data complicated the data analysis. As drop outs coincided with the impact at the seat, they may have resulted from high frequency oscillations, or velocity affecting the resolution of the system. Prior to Phase 4, further investigation was recommended to determine the optimum Optotrak parameters in order to obtain higher sampling rates without missing data.

65

Biochemistry

Biochemical measures were subject to wide inter- and intra-individual variation at rest and in response to motion exposure. Because of the variability, the small subject cohort did not provide sufficient analytical power to identify statistical significance or strong trends in the data.

As a result of individual variability, a subject identified as a "responder" may be masked by the group data. In a practical sense, a "responder" in a variable which suggests damage to skeletal muscle may be at greater risk of injury if other physical activity is attempted concurrently or immediately following exposure to repeated shocks and impacts. This was evident when individual CPK data were plotted (Figure 21). Subject 1 and 4 show a clear elevation of CPK at 12-, 24- and 36-hours post-exposure in at least one experimental condition. Subject 3 has a more variable response, and subject 2 is essentially a "non-responder". An unusually high baseline value for CPK in subject 2 in Experiment L6 suggested that the subject experienced some muscular trauma or extreme muscular exertion one or two days before the study began

The degree of elevation of CPK in the current study, while clinically significant, is small compared to that expected after severe physical exercise (Apple and Rhodes, 1988). The muscle mass under stress in this study is much more localized than during severe physical exercise. Therefore, a lower CPK value (than expected in response to intense physical activity) could still indicate a high level of local stress.

Other biochemical data did not produce statistical evidence of fatigue or tissue damage. Neither EMG nor ECG data indicated obvious fatigue, which supports the lack of a biochemical marker of fatigue (e.g., elevated lactate, ammonia, or potassium).

Factors which may have affected the interpretation of the biochemical data included: poor quality control in some biochemical analyses; high resting values in some biochemical variables; general stress associated with blood sampling; a small number of subjects with a high variability in subject characteristics; and two subjects who appeared to have had a sub-symptomatic illness based on a high white blood cell count. It is also quite likely that the exposure was not long enough or severe enough to adversely affect the subjects.

Based on the Phase 3 results, selected biochemical measures were recommended for the experimental phase. To improve the interpretation of biochemical data, it was recognized that future experiments should include:

- longer duration, higher exposure experiments to increase the likelihood of an effect;

- kidney function tests, including a measure of glomerular filtration rate before and after the experiment;

- a larger number of subjects in the long-term experiments; and

- pre-experiment blood samples to screen the subjects for any sub-symptomatic illness which confounds the data.

In addition, biochemical data obtained following "real" exposures to vehicle stress, for example in prolonged field studies, were recommended to provide further insight to the physiological stress associated with WBV and repeated shock.

Phase 3 conclusions

Results from the pilot experiments highlighted limitations in the current standards for human exposure to vibration and shocks. New standards should consider the non-linearity of the body, which may result in different response curves that are determined for each axis (x, y and z), direction (positive and negative), and for different shock magnitudes. The curves may have a totally different shape for very high magnitude, compared to less severe shocks.

At the completion of Phase 3, the following conclusions were made.

- Responses to the shocks measured as spinal acceleration, internal pressure, chest and abdominal displacement, and EMG showed similar patterns of frequency response (i.e., a dependence on the shock waveform frequency).

- Responses measured for shock frequencies of 2 to 11 Hz did not agree with transmission (weighting) curves in current standards (ISO 2631, 1982; BS 6841, 1987; ASCC, 1982).

67

- There was evidence of non-linearity in the human biodynamic response to shocks as reflected in:

  - changes in frequency of peak (transmission) response in the x and z axis with different magnitudes of shock;

  - changes in transmission ratio with different magnitudes of shock; and

  - the shape of the spinal acceleration response to individual shocks in the negative z axis.

- The magnitude of response to z axis shocks was higher than the response to shocks in the x and y axes.

- The dominant spinal acceleration and internal pressure responses to negative z axis shocks were associated with the subject hitting the seat. This response contained very high frequency components (i.e., greater than 20 Hz).

- The pilot experiments did not show conclusive evidence of fatigue induced by 2 hour exposures to repeated shock and vibration in either biochemical indicators, EMG response or ECG parameters.

- There was biochemical evidence of muscle damage in some subjects following 2-hour exposures to shocks and vibration.

- The magnitude of muscle response to shock is typically less than 10 percent of maximal voluntary contraction.

- The pattern of muscle response to shocks involves two phases: stabilization of the upper torso and re-establishment of a neutral posture.

- Performance measures induced changes in some ECG parameters.

Phase 3 recommendations

Based on the Phase 3 experiments, the following recommendations were made:

- Standards developed for exposure to vibration and repeated shocks should account for:
  - non-linearity of response;
  - differing responses to x, y, and z axis inputs; and
  - differing responses to positive and negative directions of shocks in the x and z axes.

- Further investigations of the human response to individual shocks are required, including:
  - shocks in the negative x axis and positive z axis directions;
  - shocks at low frequencies (for example, 1 to 4 Hz);
  - shocks having a higher frequency waveform (for example, greater than 20 Hz); and
  - larger magnitudes of shocks.

- Further investigation is required of exposures that are of longer duration and increased severity to more accurately simulate a typical military mission.

- More frequent recovery measures should be taken over a longer recovery period to observe possible fatigue.

- The fourth phase of the project should include the following measures:
  - acceleration at the spine;
  - displacement of the spine (measured by Optotrak);
  - internal pressure;
  - EMG;
  - ECG;
  - biochemical markers (including hydroxyproline, lactate, potassium, CPK and glomerular filtration rates);
  - subjective performance measures.

Phase 3 limitations

A number of limitations were apparent in the pilot study. These are also discussed in an overview of limitations following Phase 5. Interpretation and application of the Phase 3 results are, therefore, restricted to the limitations described herein.

The limitations recognized in Phase 3 included, but were not limited to:

- gender (all subjects were male);

- age (all subjects were aged 20 to 40);

- healthy, relatively fit, military population;

- small number of subjects;

- large inter- and intra-subject variability;

- long duration experiments were limited to 1- and 2-hour exposures, which do not reflect a typical military mission;

- a full range of shocks were not tested with respect to direction, magnitude, waveform frequency or direction (positive or negative); and

- the excursion capability of the MARS shaker table which meant that very low frequency, high amplitude shocks could not be investigated.

Phase 4: Experimental phase

Phase 4 Introduction

The pilot tests conducted in Phase 3 provided essential information for design of Phase 4 experiments. From the results, of Phase 3, motion signatures and measures to evaluate the relevant human responses were selected which could provide the required data to develop a standard for a HHA method.

Short-duration experiments were designed to evaluate further the human response to a range of individual shocks, including varying waveform frequencies, amplitudes, directions and the response to a single amplitude swept sine wave. These experiments were designed to provide information about the transmission characteristics of single shocks in the x, y and z axes. The longer duration experiments were designed to assess the potential fatigue and recovery effects of exposure to repeated shocks for up to 7 hours.

Motion exposures and the VDV

In designing the individual shock signatures for long term experiments, the VDV of BS 6841 was used to develop comparative exposures containing different shock amplitudes and rates. By using motion signatures with the equivalent VDVs, the human response to individual shocks and prolonged motion exposures could be compared according to shock frequency, amplitude, direction and rate.

According to the BS 6841, the VDV of any shock signature is proportional to the amplitude of the shocks and to the fourth root of the shock duration and number of shocks (BS 6841, 1997). The VDV is defined by the function:

$$\text{VDV} = \left(\int_0^t a_w(t)^4 dt\right)^{1/4} \qquad \text{m·s}^{-1.75} \qquad (9)$$

where $a_w(t)$ is the frequency weighted acceleration. Based on this equation, if shock amplitude is doubled when shock rate is constant, the VDV is doubled. However, to double the VDV when both shock rate and amplitude are constant, the exposure duration must be increased by a factor of 16.

71

In the BS 6841, the z axis has a higher frequency weighting factor than the x and y axes. As the purpose of experiments LT1 and LT2 was to compare the relative severity and tolerance of similar shocks in the three axes, comparative VDV values were calculated using the BS 6841 z axis weighting for all shock signatures and directions.

Phase 4 objectives and methods

The Phase 4 protocol consisted of six experiments. The first experiment examined the human response to individual shocks (designated as short term experiment: ST1). The five longer duration experiments examined human tolerance to repeated shocks (designated as long term experiments: LT1, LT2, LT3, LT4, and LT5).

The overall objective of Phase 4 was to evaluate the biomechanical, physiological and biochemical indices of injury identified during the Phase 1 literature review and Phase 3 pilot experiments which would lead to prediction of risk of injury and the development of a HHA standard. Global objectives of the Phase 4 study were further defined as they applied to short term and long term experiments.

Objectives of short term exposures

- to establish a relationship between the human response (spinal acceleration, spinal displacement, EMG, internal pressure) to shocks and shock waveform frequencies in the +x, -x, ±y, +z and -z axes.

- to compare the biomechanical response to shock frequencies and subjective ratings of shock severity to frequency weighting factors in the ISO 2631, the BS 6841, and DRI.

- to establish whether the relationship between the shock amplitude and transmission of the shock to the human is linear or non-linear.

Objectives of long term exposures

- to determine human tolerance of prolonged exposure to repeated shocks in different directions (i.e., in the +x, -x, ±y, +z and -z axes).

72

- to estimate a daily and weekly exposure limit for repeated shocks (of different magnitudes) in the +x, -x, ±y, +z and -z axes.

- to examine the effects of recovery on the human response to repeated shocks.

- to compare subjective tolerance ratings of shock exposure severity with the VDV and other predictors of fatigue or material failure.

Methods

Following subject briefing, medical screening and orientation, fifty-four male subjects participated in the Phase 4 experiments. During the experiments, subjects were exposed to a series of mechanical shock in three biodynamic axes (x, y, and z) superimposed on a background of random vibration. Exposure duration ranged from 3.75 minutes (for a single motion signature) to 7 hours.

Table 10 outlines the six experiments in Phase 4 which were designed so that subjects participating in more than one experiment did not exceed the cumulative weekly and monthly exposure limits of 20 and 30 hours respectively.

Rest breaks were included in experiments LT3, LT4 and LT5. The combination of exposure and rest break duration for the experiments are listed in Table 11.

Table 10
Phase 4 experiments

Exp.#	Experiment Duration (minutes)	# Subjects	Daily VDV $(m \cdot s^{-1.75})$
ST1	35	10	31.6
LT1	18.75	10	38.6
LT2	120	6	48.3
LT3	420	10	66.1
LT4	240	8	57.4
LT5	60/180	10	58.2
Total		54	–

ST= short term; LT= long term

Table 11
Exposure duration and rest breaks

Experiment	Exposure Duration	Rest Break Type
LT3	7 hours	two 15 minute and one 30 minute breaks per exposure
LT4	4 hours per day for 5 consecutive days	one 15 minute break per exposure and overnight between consecutive days
LT5 (Intermittent condition)	1 hour total (3.75 minute signatures separated by rest breaks)	sixteen 7.5 minute breaks for 1 hour of exposure

LT= long term

Table 12 summarizes the dependent measures for each of the Phase 4 experiments. More information on the dependent measures is summarized in Phase 3, and a detailed explanation of the methods and equipment used are provided in the Phase 4 report (Cameron et al., 1996).

Table 12
Dependent variables in Phase 4 experiments

	ST1	LT1	LT2	LT3	LT4	LT5
ECG	YES	YES	YES	YES	YES	YES
EMG	YES	NO	NO	YES	YES	NO
Acceleration	YES	NO	NO	YES	YES	NO
Optotrack	YES	NO	NO	YES	YES	NO
Internal Pressure	YES	NO	NO	NO	NO	NO
Blood	NO	NO	YES	YES	YES	NO
Urine	NO	NO	YES	YES	YES	NO
Subjective Response	YES	YES	YES	YES	YES	YES
Cognitive Performance	NO	NO	NO	YES	YES	NO
Anthropometry	YES	YES	YES	YES	YES	YES

ST=short term; LT= long term.

Biological specimens (blood and urine) were collected
and analyzed in three of the long duration experiments
(LT3 and LT4) and the two exposures in LT2 which included
4 g shocks. These were examined for evidence of the following:

- muscle damage;
- fluid shift;
- blood clotting;
- glucose in blood and urine;
- fatigue;
- inflammation;
- bone stress and remodeling; and
- kidney, bladder, or urinary tract dysfunction.

Phase 4 results and discussion

Skin transfer function

Results from Phase 3 highlighted the requirement for more
extensive analysis of the skin transfer function. Hence, prior
to analysis of acceleration data in Phase 4, new approaches to
modeling of the bone-to-skin transfer function were investigated.
Additional mathematical processing was used to determine, and
correct for, any movement of the skin surface relative to the
underlying bone (spinous process). This correction required
a knowledge of the bone-to-skin transfer function for the y and
z spinal accelerometers for each subject. Measured acceleration
signals were then multiplied by their respective inverse transfer
functions. This correction eliminates any contribution of bone-
skin movement and provides the true acceleration at the spinous
process. As x axis accelerometers measured motion perpendicular
to the skin surface, they were not sensitive to shearing motion
between the spinous process and the skin. Hence a skin transfer
function was not computed for the x axis.

Linear modeling approaches for identifying the transfer function
of the skin

y axis skin transfer function

Only low frequency accelerations (less than 20 Hz) were
recorded at the y axis spinal accelerometers in response
to shocks at the seat. Skin perturbation data collected

in the y axis were therefore low pass filtered, and the tissue-accelerometer subsystem was then modeled as a simple Kelvin element.

To estimate the acceleration response of the vertebra underlying the accelerometer, y axis spinal acceleration data of each experimental exposure were converted from the time domain to the frequency domain using a forward FFT. The frequency spectrum was multiplied by the inverse of the bone-to-skin transfer function, and the data then reconstructed in the time domain using an inverse FFT. This mathematical treatment of the data provided an estimate of the input acceleration signal at the spinous process necessary to produce the output acceleration signal measured at the skin surface.

z axis skin transfer function

Skin perturbation data showed that the free response of the vertebra-skin subsystem contained both high and low frequency components in the z axis acceleration (Figure 22). Hence the system could not be truly represented as a simple Kelvin element (i.e., a single degree of freedom, second order system).

Analysis of spinal accelerations in the z axis revealed substantial acceleration "spikes" in response to shocks input at the seat. These acceleration spikes occurred in response to 2, 3 and 4 g shocks and contained frequency components above 20 Hz. The higher frequency responses (in the range 20 to 150 Hz) were most noticeable as a result of the 4 to 8 Hz shock inputs, and were present in response to both positive and negative shock directions.

Acceleration spikes tended to coincide with the subject hitting the seat. Therefore, they could not be considered to be artifacts in the data which could be removed by low pass filtering. Hence, the assumption of a simple Kelvin element in determining the z axis skin transfer function was inadequate.

When this model was applied (using the method described for the y axis), the inverse transfer function resulted in an artificial magnification of the high frequency components of the accelerometer signal. Theoretically, these high frequencies would not have been transmitted if the second order linear model was correct. To circumvent this problem, new approaches to modeling of the bone-to-skin transfer function were investigated. These methods included parametric modeling using Least Squares

Estimation, Prony's algorithm (Parks and Burrus, 1987) and Steiglitz-McBride (STMCB) iteration (Steiglitz and McBride, 1965); and a linear approximation of a two degrees of freedom model.

The z axis spinal acceleration data were separated into low frequency and high frequency components. Each component was treated separately with a linear correction, and then the two components were summed to obtain the corrected acceleration at the vertebra. The spinal acceleration data recorded by the L4 accelerometer in response to a negative 4 g, z axis shock at the seat is shown in Figure 23. For comparison, the predicted acceleration at the spinous process after correction by the compensation filter is superimposed on the acceleration data.

Spinal acceleration response to individual shocks

x axis spinal acceleration

In the response to x axis shocks at the seat, the acceleration response curves for each shock direction and amplitude showed a similar curvilinear relationship with shock frequency. The transmission ratios were highest at 2 to 4 Hz, and typically declined in a curvilinear manner with increasing frequency to 20 Hz. Although similar in nature, the transmission curves at each shock amplitude were not identical. The mean transmission ratios of all amplitudes of x axis shocks were generally higher at L2 than at T1. This effect is shown in Figure 24 and Figure 25.

Mean transmission ratios at L2 were generally greater in the positive x axis than in the negative x axis. A similar effect was also evident in the thoracic transmission ratios in response to shocks at low frequencies (4 to 6 Hz), but was not evident at higher frequencies up to 20 Hz. Comparative results are shown in Figure 26.

y axis spinal acceleration

As the body is symmetrical in the sagittal plane, acceleration data were collected only in the positive y axis. In response to each shock amplitude, the lumbar (L3) transmission curves showed a relationship with shock frequency that was similar to the x axis. Transmission ratios were highest at the lower frequencies (2 to 5 Hz) and declined in a curvilinear manner with increasing frequency up to 20 Hz (Figure 27). The 4 g shocks showed a peak transmission ratio at 5 Hz, but this

77

effect was not evident at the other shock amplitudes. Comparable transmission data were observed at T2. The 4 g shocks produced the highest transmission ratios at low frequencies, and again showed the greatest attenuation at the higher shock frequencies (11 to 20 Hz).

The mean transmission ratios of all shock amplitudes indicated that the transmission ratios were consistently higher at the lumbar (L3) compared to the thoracic (T2) level, particularly at the higher shock frequencies. This effect, shown in Figure 28, was more pronounced in the y axis than shown in the x axis data (Figure 24).

z axis spinal acceleration

Although there were distinct differences in the L4 and T3 transmission curves in response to shock amplitude in the z axis, some similarities were apparent at both locations and in response to both positive and negative input (Figure 29 and Figure 30). Transmission ratios were highest at the low shock frequencies (2 to 4 Hz) and declined in a curvilinear manner with increasing frequency to 20 Hz.

Examination of the mean transmission ratios of all amplitudes of positive z axis shocks indicated that the transmission ratios were similar at both the lumbar (L4) and thoracic (T3) levels. The only exception to this pattern was the transmission of 4 g shocks at frequencies of 2 to 6 Hz, where the thoracic transmission ratio exceeded the lumbar transmission ratio. The transmission ratios of negative z axis shocks were similar at the lumbar and thoracic levels. The L4 and T3 transmission ratios of 3 g and 4 g shocks were much higher than those of 0.5 g and 1 g shocks, particularly in the range of 4 to 8 Hz. For a shock input of 4 Hz at the seat, a T3 transmission ratio of 0.9 was obtained for a 1 g shock, whereas, the transmission ratio increased to 2.6 in response to a 4 g shock.

At low frequencies (2 to 6 Hz), the lumbar transmission ratios were generally greater in response to negative z axis shocks than to those in the positive z axis. (Figure 31 and Figure 32). This tendency was reversed at the high frequencies (11 to 20 Hz) where the lumbar transmission in the positive direction was greater than in the negative direction. There was a clear amplitude effect in the transmission ratios of both positive and negative z axis shocks. This effect was much greater than noted in either the x axis or y axis data.

Second component of spinal acceleration response to z axis shocks
at the seat

Figure 33 illustrates the two-component response of a
positive 4 g, 4 Hz seat shock measured at the lumbar spine. The
two compontent response is seen in both the positive and negative
z axis. The initial response to the input shock causes the
subject to briefly leave the seat. This is followed by a second
response as the subject impacts the seat. The impact of the
subject on the seat is also recorded in the seat acceleration
signal as a high frequency pulse approximately 0.3 seconds after
the initial shock peak.

An example of the mean transmission ratios of the second
identified response to 2, 3, and 4 g, z axis shocks applied to
the seat is illustrated in Figure 34. The second acceleration
response to positive z axis shocks had transmission ratios
similar to the initial acceleration response for shocks at 4 Hz
(transmission ratios of 1.5 to 3 in both the first and second
response). In contrast, the negative z axis shocks produced
a second acceleration response that was less than the first
response for both lumbar and thoracic acceleration (transmission
ratios of 2.5 to 5 for the first response and 0.3 to 0.8 for the
second response). For z axis shock frequencies above 4 Hz,
transmission ratios for the second response were progressively
less than the transmission ratios computed for the first
response. The lumbar and thoracic transmission ratios of the
second response declined rapidly as frequency increased, reaching
nearly zero transmission by 11 Hz.

High frequency spikes

The results clearly establish the presence of high frequency
acceleration spikes at both the lumbar and thoracic level in
response to larger amplitude shocks (2 to 4 g at a frequency of
4 to 8 Hz). As the nature of these high frequency spikes had
not been reported previously in the literature, these effects
were unexpected (in terms of acceleration magnitude and frequency
content). Interpretation of the data, in particular with respect
to skin movement effects, has to be regarded as tentative.

A comparison is provided of the shock transmission ratios
obtained from the raw acceleration data, the data corrected for
bone-skin transfer effects, and data which have been low pass
filtered at 40 Hz (Figure 35). Although application of the skin
transfer function and 40 Hz filter resulted in a considerable

79

attenuation of the recorded spinal acceleration, the transmission ratios exceeded those predicted by existing models.

Based on the raw data and subsequent analyses of spinal transmission, the high frequency acceleration spikes were not considered to represent either skin artifact or accelerometer measurement error. For high amplitude shocks, there was evidence of transmission of high frequency acceleration spikes within the vertebral column which were not present in lower amplitude vibration. The correction technique for bone-to-skin transfer reported by Hinz et al. (1988), Smeathers (1989), and Kitazaki and Griffin (1995) are inadequate in these circumstances and grossly amplify the high frequency components measured. Therefore, an alternate correction technique was developed for this study.

Comparison of the spinal transmission curves to existing standards and models

The mean transmission curves for spinal acceleration in response to x and y shocks were compared to the following standards and models:

x axis   frequency response curves of the BS 6841 x axis filter (1987) and the DRI (10 Hz) model for the x axis (Payne, 1984) (Figure 36).

y axis   frequency response curves of the BS 6841 y axis filter (1987) and the DRI (7.2 Hz) model for the y axis (Payne, 1984) (Figure 37).

For x axis shocks, the DRI (10 Hz) response curve consistently overestimated the magnitude of acceleration transmitted to the spine by 2 to 3 fold. The natural frequency of the DRI (10 Hz) model was also much higher than suggested by the spinal response data in the study. A better approximation of the current data was achieved by the output of the BS 6841 filter. However this filter consistently produced a slight overestimation of shock transmission in both positive and negative directions at the lumbar and thoracic levels.

In response to y axis shocks, the DRI (7.2 Hz) model response curve overestimated several fold the amplitude of accelerations transmitted to the spine. The natural frequency of the DRI model (7.2 Hz) was also much higher than suggested by the spinal transmission data. A better approximation of the

transmission curves was achieved by the output of the BS 6841
filter. However, this filter underestimated shock transmission
at the lumbar level for low frequency shocks (2 to 6 Hz), and had
a slower decay rate with increasing shock frequency than the y
axis spinal transmission data.

Due to the amplitude effect in the z axis response, it was
not considered meaningful to compare the mean response curves of
all shock frequencies with existing standards. Thus, in response
to 1 g and 4 g z axis shocks, the individual transmission curves
were compared to the output of the following biodynamic models
and filters contained in current standards:

z axis: frequency response curves of the BS 6841 (1987)
             z axis filter, the Fairley-Griffin model
             (Fairley and Griffin, 1989), the DRI (8.4 Hz)
             model contained in the ASCC (1982). For 4 g
             shocks only, the data were also compared to the
             revised DRI (11.9 Hz) model of Payne (1991).

In response to 4 g shocks, all four models clearly
underestimated transmission effects at the spine (Figure 38).
In response to 1 g shocks at the seat, both the BS 6841 filter
and the Fairley-Griffin model underestimated the transmission
of 1 g shocks (Figure 39). Although the DRI (8.4 Hz) model
approximated the amplitude of transmission to 1 g shocks
over part of the frequency range, the DRI (8.4 Hz) response
showed a distinctly different relationship with shock frequency
than the response measured at L4.

Linear versus non-linear models in standards and guidelines for
shock transmission

The results of transmission of spinal acceleration clearly
illustrate the non-linear relationship between an input shock at
the seat and the acceleration response at the spine. In general,
the effect of shock frequency on transmission ratio is stronger
as shock amplitude increases. Therefore, the existing biodynamic
models and guidelines which have linear characteristics
(i.e., the BS 6841 (1987) and DRI from the ASCC standard (1982))
will be valid for only a limited range of shock amplitudes and
shock frequencies. The non-linearities identified in the current
experiments indicate that existing linear models and weighting
filters will incorrectly estimate the transmission of large
amplitude shocks, particularly at low frequencies. The linear
filter, BS 6841 (1987), underestimates the transmission ratios
for all axes, whereas the DRI linear model overestimates the

81

transmission ratios for the x and y axes, but underestimates for the z axis.

The transmission curves generated from Phase 4 experiments supported the need for development of a predictive model that is both non-linear with shock amplitude and sensitive to shock direction and axis.

Optotrak

Data for both spinal acceleration (accelerometers) and displacement (Optotrak) were collected for two reasons. Displacement data were required to examine posture and for use in Phase 5 biomechanical modeling; acceleration data were required for the calculation of shock transmission. Displacement data (Optotrak) collected during Phase 4 experiments were analyzed during Phase 5 to provide detailed information about spinal motion for implementation in a biomechanical model to estimate stress in the spine.

Internal pressure

Internal pressure response to x, y and z axis shocks

The internal pressure response to x axis shocks was non-linear with shock amplitude, particularly at frequencies less than 8 Hz. Response ratios were highest at 2 to 4 Hz and declined in a curvilinear manner with increasing frequency to 20 Hz. The internal pressure response to y axis shocks was also non-linear, although the response curves of each input amplitude showed some variation in the pattern with respect to shock frequency. The peak response ratio for each amplitude was observed at the lowest frequency measured. Response ratios for 2 g, 3 g, and 4 g shocks were highest at 4 Hz and declined in a curvilinear manner with increasing frequency to 20 Hz.

Consistent with x and y axis data, internal pressure response ratios to z axis shocks were greatest at the lowest frequency measured and declined in a curvilinear manner with increasing frequency (Figure 40). There was very little change in response as frequency increased from 11 Hz to 20 Hz. There was a clear amplitude effect on internal pressure response ratios for frequencies of 8 Hz and below. At these frequencies, response ratio increased with shock amplitude.

Second internal pressure response to z axis shocks

Two-components were observed in the internal pressure response to z axis shocks. As was indicated for the spinal acceleration, the initial response was a result of the shock input at the seat that caused the subject to briefly leave the seat, and the second response corresponded to the subject impacting the seat.

An example of the mean internal pressure response ratios of the second response event for 2, 3 and 4 g, z axis shocks is illustrated in Figure 41. The response ratios followed a pattern similar to the acceleration transmission response at L4, with a maxima at 4 Hz and approaching zero response at 11 Hz. The second response ratio was greater for positive than negative shocks. The initial response to shocks was greater than the second response for both positive and negative directions. For example, a negative z axis 4 Hz, 4 g shock produced an initial response ratio of 6.3 and a second response of 1.0.

Overall internal pressure response

Phase 4 experiments characterized the frequency- and amplitude-dependence of the internal pressure response to shocks applied at the seat. As with acceleration transmission in the spine, the internal pressure response was non-linear with amplitude for shock frequencies below 11 Hz.

The measured internal pressure response to shocks at the seat is likely to result from a combination of internal events. Co-activation of abdominal and back muscles in response to a shock was observed in the EMG data in Phase 3. This co-activation, along with activity of the diaphragm, will increase intra-abdominal pressure. In addition, internal pressure was measured in the colon at the base of the abdomen. Hence, the motion of organs and tissues positioned superior to the pressure transducer will influence the locally measured pressure. Downward motion of abdominal organs will exert a force on the lower colon, which will be recorded as a transient increase in internal pressure.

Phase 3 experiments demonstrated that the internal pressure response to a 3 g, z axis shock could exceed the maximal voluntary pressure that subjects could produce (greater than 200 mm Hg). Such large and relatively long lasting pressure transients in the abdomen may provide a counter-force to the

83

inertial moment of the upper torso and head. If this is true, the internal pressure response may reduce axial loading of spinal elements by providing an alternate pathway for load transmission, and reduce bending moments.

Based on these data, a biomechanical model designed to estimate stress in the spine in response to low frequency shocks should include the influence of internal pressure. If internal pressure significantly affects the estimates of spinal stress, development of a non-linear predictive model to estimate internal pressure in response to seat shocks would enhance the HHA method. The response curves suggest that high amplitude, low frequency (less than 11 Hz) shocks present the greatest risk of injury to the organ systems.

Electromyography

Long duration experiments (7 hour exposure or 4 hours per day for 5 consecutive days) were expected to result in fatigue of back muscles. This was of interest for the HHA method because of possible association between back muscle fatigue and chronic low back pain, diminished functional capabilities of the individual, and increased stress on passive tissues of the back.

Back muscles are partially responsible for the maintenance of posture during motion, especially in a seated position. This may be critical for prevention of injury caused by a soldier hitting instrumentation or walls inside a vehicle. Muscle fatigue may also reduce a soldier's capacity to perform physical tasks immediately after prolonged motion exposure, particularly if those tasks involve extensive recruitment of back muscles. Thus, a soldier might be at higher risk of injury due to operational activities if physical tasks are preceded by prolonged travel in TGVs.

Muscle fatigue is believed to be a contributing factor in the etiology of chronic low back pain (Roy, DeLuca and Casavant, 1989), although the mechanism of this association is not well understood. It has also been suggested that the progression of muscle insufficiency or fatigue leads to increased stress on the passive tissues of the spine (Bogduk, 1984; Gracovetsky, 1988). Muscle fatigue, therefore, may have multiple consequences that are relevant to the health of the soldier.

84

Mean frequency

Approximately twenty-five percent of subjects demonstrated a change in mean frequency (MF) of EMG during test contractions after motion exposure in this study. However, this change was equally likely to be an increase or decrease in MF. Although the classical literature argues that muscle fatigue results in a decreased MF, recent research has also identified an increase in MF of back muscle EMG associated with fatigue (Voss and Krogh-Lund, 1989).

In the present experiments there was no relationship between the change in MF and subjective reporting of discomfort, estimated tolerance time, or the time of exposure termination. Individuals who reported a great degree of discomfort, or had experiments terminated early did not demonstrate a greater probability of showing a change in MF than subjects who completed the full experimental duration. Similarly, there was no relationship between the magnitude of change in rms EMG and any of these factors, including total experiment duration.

RMS EMG

In experiment LT3 (up to a 7 hour exposure) there was an increase in rms EMG activity in the last measurement interval compared with the first measurement interval at all muscle sites recorded on the back. There was an increase in rms EMG of forty percent, on average, with a group mean rms EMG of 0.20 volts in the first sampling trial and 0.28 volts in the last sampling trial.

EMG and fatigue

The development of measurable, enduring muscle fatigue was not demonstrated as a consistent result of exposure to relatively severe motion for up to seven hours in one day, or for up to five consecutive days of four hours per day. However, an increase in rms EMG activity was measured consistently in all subjects during motion exposure of two and one half hours to seven hours (LT3).

The increased rms EMG activity may indicate a reduced capacity or increased effort to exert control over posture during motion. However, the magnitude of EMG response to a typical shock remained well below the level of a maximal voluntary contraction. Muscle fatigue was not related to discomfort, subjective reporting of back pain, or diminished functional

85

capacity in the generation of a contraction at twenty percent of a maximal voluntary contraction.

Biochemistry

In Phase 4 experiments, the consistent absence of a detectable biochemical change in blood and urine variables was unexpected. Based on the results of Phase 3, coupled with the increased exposure intensity (i.e., higher VDV) and longer duration exposures, an indication of fatigue, stress or injury was anticipated. Although the subject numbers were large enough to provide sufficient analytical power in relation to the expected changes in biochemical variables, neither statisical significance nor strong trends in the data were identified.

Irregular changes in CPK and LDH concentration in some subjects following motion exposure, as well as clinically elevated CPK in some pre-exposure measurements, suggested that subjects did not strictly follow the repeated instruction to eliminate physical exercise for the duration of the study.

Glomerular filtration, evaluated by creatinine clearance measurements, required a timed (24 hour) collection of urine. In this study, subjects were relied upon to perform the collection. Because some 24 hour urine volumes were smaller than the normal daily minimum (as low as 190 ml in 24 hours) it was strongly suspected that some urine volume was lost. Inaccurate urine volumes affected calculated creatinine clearance. Hence, the mean creatinine clearance data are suspect. As well, some subjects did not return their urine collection containers which resulted in missing data.

Although disappointing, the lack of a clear biochemical marker of stress, acute or persistent fatigue, or tissue damage is consistent with other measures in the study. EMG data did not indicate obvious fatigue, and subjects did not report severe discomfort or injury in their subjective responses. There was no evidence of hypoglycemia in these experiments, although the LT2 experiments (i.e., 2 hour exposure duration) were too short to expect a large reduction in blood glucose, and during 4 and 7 hour exposures, subjects were able to eat during scheduled rest breaks which were no more than 2 hours apart.

Several factors which affected the interpretation of the biochemical data included: loss of samples due to hemolysis or clotting of blood; loss of urine volume in 24 hour urine collections; high concentrations in some pre-exposure measurements which were not elevated in baseline measurements; general stress associated with blood sampling; high variability in subject characteristics; and several subjects who, based on LDH and CPK data, appeared to have participated in intense physical activity. Because of the safety precautions observed in the design of the experiments, and the physical limitations of the MARS facility, is also likely that the exposures were not long enough or severe enough to affect the subjects in a way that could be measured through blood and urine. Even if the higher intensity shocks resulted in local tissue or structural trauma, the resulting biochemical changes in blood or urine may have been too small to be detected by current analytical techniques.

Physical status and body part discomfort

Although there was no evidence of biomechanical damage or serious muscular fatigue in the quantitative data, clear effects of discomfort, pain and stiffness lasting 24 to 72 hours beyond exposure were apparent in subjective data, subject debriefing and observation of the subjects.

Body part discomfort reported by subjects during motion exposure, at rest breaks, and in conjunction with blood and urine sampling, was most often associated with the neck (C7 to T3), between the scapulae (T6 to T9), at the lumbar spine (L1 to L3) and buttocks. Subjects reported discomfort as "tightness", "numbness", "throbbing", or "pain" in the muscles and occasionally in the spine. A few subjects reported headaches, which disappeared shortly after exposure (within one hour). Generally, the type of physical discomfort experienced post-exposure was comparable to that experienced from strenuous physical activity, for example lifting weights. However, the onset of soreness was more rapid with exposure to repeated shocks than usually associated with physical activity. For example muscular soreness, which is typically greatest two days after a weight lifting session, developed within 24 hours after a 4 hour exposure on the MARS.

The symptoms of soreness, pain and post exposure muscle stiffness may also be due to postural fatigue, caused either

by increased muscle tone or sustained contraction in response to shocks. However, evidence strongly suggests that some localized pain and stiffness could be derived from tissue stresses other than muscle. For example, irritation to ligamentous, connective tissues and possibly facet joint or discs may result in similar symptoms. Some subjects reported bilateral pain over the erector spinae muscles, whereas others reported pain more central to the spinal column and located directly over specific vertebrae. Several subjects noted severe soreness of the coccyx which warranted treatment with analgesic and anti-inflammatory drugs. This suggests that the passive tissues (i.e., bone, ligament, tendon, cartilage) may be at greater risk of injury than muscle.

Subjective response to single shocks

In short term experiments, the subjective response to single shocks was examined to determine the most severe characteristics of motion exposure, to provide a relationship between subjective response and spinal transmission of shocks, and to examine the relationship between existing biodynamic models and subjective response. In Phase 5, knowledge of these characteristics and relationships will contribute to the development of a HHA method related to exposure to mechanical shocks.

Effect of motion characteristics

Subjective severity ratings demonstrated trends in response to different motion characteristics including shock frequency, axis and direction. For all shock conditions, the lowest tested shock frequency resulted in the highest mean severity rating, which decreased in a curvilinear manner with increasing shock frequency. This is shown in Figure 42 which compares the subjective severity ratings for 0.5 g to 4.0 g z axis shocks between 2 to 20 Hz.

The mean rating values in the different axes demonstrated that, for the same amplitude and frequency, z axis shocks were rated as significantly more severe than x or y axis shocks. The mean severity ratings for x and y axis shocks were not significantly different. No significant difference was observed between severity ratings to positive and negative shocks in the x and z axes.

If subjective severity is incorporated into a health hazard standard, low frequency z axis shocks should be rated as the most

hazardous motion condition. Both the x and y axes shocks should be weighted less than z axis shocks. Weighting factors for all axes should decrease as shock frequency increases.

Linearity of subjective severity to shock amplitude

Across the tested shock frequencies (2 to 20 Hz), a non-linear amplitude effect was demonstrated for subjective severity for all shock axes and directions. Similar non-linear amplitude effects were evident in the spinal transmission responses. By comparing normalized severity ratings, low frequency (below 8 Hz), high amplitude shocks were rated relatively more severe than low amplitude shocks (Figure 43). At high frequencies (above 11 Hz), low amplitude shocks were rated relatively more severe than high amplitude shocks.

To accurately model severity of shocks in any exposure standard, the frequency weighting function would need to account for the effect of amplitude. Thus, the development of a non-linear model is required.

Relationship between subjective severity and biodynamic model outputs

Subjective severity ratings were compared to the normalized output of existing biodynamic models (the BS 6841, the Fairley-Griffin model, and the DRI 8.4 Hz and 11 Hz models). In response to z axis shocks, both frequency and amplitude dependent effects were observed. Each of these models underestimated subjective severity at low frequencies, and overestimated severity at high frequencies. The cross-over point from underestimating to overestimating severity for all models, ranged from 4 to 15 Hz, and was dependent on shock amplitude

The closeness of the relationship between severity ratings and the existing biodynamic models decreased progressively in the following order: Fairley-Griffin model, DRI model (8.4 Hz version), DRI model (11.9 Hz version) and BS 6841 filter. Figure 44 compares the subjective severity rating to the output from the Fairley-Griffin model for positive z axis shocks.

Comparison of subjective severity ratings and normalized output acceleration of biodynamic models in response to x and y axis shocks demonstrated similar trends to those shown for the z axis. The closeness of the relationship between severity ratings and the models was higher for the BS 6841 filter than for the DRI model for both x and y axes.

Overall, existing biodynamic models which were tested underestimated subjective severity at low frequency shocks, and overestimated severity at higher frequencies. The inaccuracy of the models was dependent on input shock amplitude. In this study, both subjective severity and spinal transmission exhibited a non-linear amplitude effect. However, the existing biodynamic models and filters to which both subjective and spinal transmission data were compared are based on the assumption that the human response to motion input is linear with increasing amplitude. As demonstrated by the non-linear effects exhibited in this study, it is unlikely that existing biodynamic models can account for the non-linear effect of amplitude observed in both subjective severity and spinal transmission.

Relationship between subjective severity and spinal transmission

Subjective severity accurately represented acceleration transmitted to the thoracic and lumbar spine for all shock axes, directions and vertebral levels. Subjective severity was more closely related to spinal transmission than to any of the existing biodynamic models and filters.

This observation may be due to the common non-linear amplitude effect observed in both subjective severity and spinal transmission. In terms of evaluating health hazard effects, subjective severity may be a valid method of estimating the spinal acceleration transmitted to the thoracic and lumbar vertebral levels.

Subjective response to long term experiments

Effect of shock axis, direction, amplitude and rate

In long term experiments, no significant differences were demonstrated in the subjective response between positive and negative shock exposures for both the x and z axes. Motion exposure in the z axis was rated as significantly less comfortable, less tolerable and more severe than in the x and y axes. Different combinations of shock rate and amplitude, with exposures of equal VDV, had no significant effect on subjective response.

90

These results indicated that in the context of subjective response, positive and negative shocks can be weighted equally, and that z axis shocks should have a higher weighting factor than shocks in either the x or y axes. These findings are supported by the frequency weighting and repeated shock evaluation methods (i.e., the VDV) presently outlined by the BS 6841 (1987).

Effect of exposure duration on subjective response

Subjective ratings demonstrated duration-dependent trends with varying shock exposure conditions (i.e., shock axis, amplitude and shock rate) and duration up to 7 hours. Duration-dependent trends included: decreased comfort; decreased predicted tolerance; increased tiredness; and increased severity.

Figure 45 illustrates subjective severity ratings as a function of duration of exposure for experiments with 4 hours exposure per day for five consecutive days. Subjective ratings for comfort, tiredness and severity in response to repeated shock exposures were dependent upon exposure duration within a daily exposure. Results of weekly exposures (LT4) suggested that the effect of daily exposure was not cumulative over the course of five days.

In long term experiments, predicted tolerance was not dependent on exposure duration. The lack of change in predicted tolerance ratings from first to last measurement intervals indicated that the predicted tolerance times provided by subjects in the first 3.75 minutes of exposure are representative of their predicted tolerance at later times in the exposure.

Effect of rest breaks on subjective response

Short rest breaks (15 and 30 minute) in LT3 and LT4 experiments did not have a significant effect on subjective ratings of predicted tolerance, tiredness or severity to repeated shock exposures. Comfort ratings to shock exposures were significantly affected by short rest breaks in LT3. In LT4, improved trends were evident in comfort ratings following short rest breaks. In addition, comfort ratings in LT5 showed the greatest difference between continuous and intermittent exposures, compared to the other subjective ratings.

Short breaks appeared to temporarily relieve the discomfort experienced by exposure to mechanical shocks. These findings were supported by the subjective comments regarding physical status, which showed that short term rest breaks relieved

91

discomfort by allowing the subject to stretch, improve blood circulation, relieve postural discomfort and provide a mental break from the constant motion.

Even though breaks were beneficial to the mental and physical state of the subject, these results do not support fully the inclusion of a short term recovery function in the development of the HHA method. Although short rest breaks did not affect all subjective response ratings at the tested shock exposure intensity, they may be more effective when exposures are more severe. As well, the duration of the rest breaks may need to be longer to allow recovery processes to have a significant effect.

During LT4 experiments (i.e., five consecutive days of 4 hour motion exposures), overnight recovery was evident from the significant decrease observed between the ratings at the last measurement interval on one day and the initial ratings on the following day. Although there was no increase in the absolute daily level of tiredness or severity ratings throughout the week, overnight breaks appeared to return the subjects to the same subjective level before each daily exposure.

Comparison of the VDV and subjective response

In the long term experiments, the ability of the VDV to predict the subjective response to an increase in shock amplitude was supported. The change in subjective comfort and severity ratings resulting from a 2-fold increase of shock amplitude (which doubled the VDV) reflected the corresponding increase in the VDV. In LT5 experiments, continuous and intermittent shock exposures having equal VDV did not have significantly different subjective ratings.

In contrast, the relationship between shock amplitude and exposure duration described by the VDV was not supported by predicted tolerance ratings. According to the VDV, the predicted exposure time would have to be reduced by a factor of 16 for an equivalent exposure dose which had a two-fold increase in shock amplitude. However, when the VDV was doubled, predicted tolerance times only increased by a factor of 2 to 5 for the different axes. Therefore, if predicted tolerance is a valid method to assess the effects of exposure dose, the use of VDV is not supported. Conversely if the VDV is valid, predicted tolerance is not.

The time dependence of the VDV matched subjective tiredness ratings for up to 4.5 hours. The lack of a significant difference between subjective ratings to continuous and intermittent exposure conditions with an equivalent VDV (in experiment LT5) also supported the concept that the VDV is able to evaluate motion exposure without concern for the acceleration-time history (i.e., inclusion of rest breaks). The ability of the VDV to accurately evaluate motion exposure was previously reported with lower amplitude exposures of much shorter duration by Griffin and Whitham (1980), Hoddinott (1986), Hall (1987), Wikström, Kjellberg, and Dallner (1990), and Howarth and Griffin (1991).

Synthetic work environment

The prolonged exposure to the motion environment, coupled with fatigue, was expected to influence performance on synthetic work (SynWork) tasks. The composite scores from six subjects during each of two SynWork trials performed during 2 hour motion exposure (LT2) are summarized in Table 13. Composite scores were consistently worse for the motion signature with negative 2 g shocks at a rate of 32 shocks per minute in the z axis than for other conditions. Composite scores were also lower for all conditions with 2 g shocks at 32 shock per minute compared to conditions with 4 g shocks at 2 shocks per minute, although these differences were not significant.

Composite SynWork scores improved progressively throughout the motion exposures that lasted up to 7 hours per day or 4 hours per day for 5 consecutive days (LT3 and LT4). The improvement was observed even though each subject completed ten to fifteen training trials prior to the experiment. However, only two out of ten subjects completed the full seven hour experiment due to predicted tolerance time in experiment LT3. A similar trend was observed for the scores of eight subjects in LT4 with cumulative exposure duration of 4 hours per day for 5 days.

Table 13
Composite scores in the synthetic work in experiment LT2

Motion Signature	1st Trial		2nd Trial	
	Mean	SD	Mean	SD
2 g -x axis, 32 shock·min^{-1}	1351	405	1496	289
2 g -z axis, 32 shock·min^{-1}	1093	335	1085	329
2 g x, y, z axis, 32 shock·min^{-1}	1266	462	1551	394
4 g -x axis, 2 shock·min^{-1}	1474	415	1655	459
4 g +z axis, 2 shock·min^{-1}	1572	312	1656	424

SD = standard deviation

Phase 4 conclusions

This was the first comprehensive study of long duration
exposure to high levels of mechanical shock and repeated impact.
Given the different effects of axis and direction on human
tolerance, and the dependence on shock magnitude and frequency,
limits developed from these data must be considered as a
guideline for the development of the HHA method. Further
investigation may be required to validate these predictions.
Conclusions and recommendations based on short and long duration
experiments are outlined below.

Short duration experiments (ST1)

- Spinal response characteristics to shocks are dependent
  on the axis, direction and amplitude of input at
  the seat.

- There is a non-linear amplitude effect for the spinal
  response to shocks which are input at the seat. The
  non-linear amplitude effect is more pronounced in the
  z axis than in the x and y axes.

- Low frequency shocks (2 to 8 Hz) are more severe than
  high frequency shocks.

- Internal pressure measurements in response to shock inputs at the seat exhibit a frequency-dependence similar to that observed for spinal acceleration which is more pronounced in the z than in the x and y axes.

- Spinal response to low frequency input at the seat (4 to 8 Hz) results in acceleration output at the spine containing high frequency (20 to 150 Hz) components which are associated with the subject hitting the seat.

- For all axes, subjective ratings of the severity of shocks at the seat show a high correlation with spinal acceleration.

Long duration experiments (LT1 to LT5)

- EMG data from muscles of the back exhibit consistent evidence of local muscle fatigue during motion exposure as brief as 2.5 hours in duration.

- There is no evidence of cumulative fatigue or trauma resulting from motion exposures in EMG, biochemistry or subjective data, after either a single 7 hour exposure or five consecutive daily exposures of 4 hours each. Although there is no biochemical, EMG or subjective evidence of fatigue, subjects report pain and discomfort during and up to 72 hours after ride exposure.

- Rest breaks, including overnight recovery, temporarily improve the subjective rating of comfort, but do not change the predicted tolerance to motion exposures with the same VDV.

- Subjects who volunteered for these experiments could tolerate a daily exposure in excess of the recommended daily dose of 15 (British Standard VDV). Some subjects are able to tolerate a VDV of 66 over a 7 hour period, or a VDV of 60 per day over a five day period.

- Short term predictions of tolerance to motion exposure are consistent with those made at longer durations.

Overall

- Existing biodynamic models (i.e., the BS 6841 and DRI), which are based on linear weighting factors for amplitude, overestimate or underestimate spinal

transmission and predicted subjective tolerance depending on the frequency of the shocks in the range of 0.5 to 4 g.

- The VDV is able to estimate the effect of shock exposures on the subjective rating of comfort, severity, and tiredness.

Phase 4 recommendations

1. New hazard guidelines for human exposure to repeated mechanical shocks need to be developed.

2. These guidelines need to include:

   - biomechanical response curves for seat-vertebrae transmission;

   - severe discomfort guidelines;

   - hourly, daily and weekly tolerance guidelines; and

   - health hazard guidelines.

3. A new health hazard standard for the human response to shocks should include a non-linear model for spinal transmission.

4. Further investigation of biomechanical effects of shocks is required.

5. Further investigation of dose-response relationships and dose-recovery relationships are required.

Phase 4 limitations

There were several limitations apparent in the Phase 4 experiments which affect the interpretation of the results and conclusions of this study. The limitations are also discussed in an overview of limitations following Phase 5.

In·Phase 4, limitations included, but were not limited to:

96

- gender (all subjects were male);

- age (all subjects were aged 19 to 40);

- healthy, relatively fit, military population;

- small number of subjects;

- inter- and intra-subject variability;

- subjects were exposed to a series of motion exposures with a fixed range of amplitude (0.5 g to 4 g) and frequency (2 Hz to 20 Hz) in positive and negative x, y, and z axes; and

- the excursion capability of the MARS shaker table which meant that very low frequency, high amplitude shocks could not be investigated.

Phase 5: Recommendations for a health hazard assessment method

Phase 5 Introduction

Phase 5 was concerned with the development of a health
hazard assessment (HHA) method and the required mathematical
models to predict the human response and risk of injury from
exposure to repeated mechanical shocks in Army vehicles. The
operation of modern TGVs over rough terrain produces repetitive
mechanical shocks which are transmitted to the soldier primarily
through the seat. Repetitive shocks from military vehicles are
typically low frequency (2 to 20 Hz) waveforms with amplitudes up
to 5 g (49 m·s^{-2}) and are separated by approximately 0.25 seconds
or more (Roddan et al., 1995; U.S. Army Health Hazard Assessor's
Guide, 1996). Seat and human response data obtained during the
Phase 4 experiments were representative of the motion environment
of military vehicles, and were utilized during model development
in Phase 5. The proposed HHA method allows for the evaluation of
repeated mechanical shock exposure and assignment of a Risk
Assessment Code (RAC) according to the HHA Program (AR 40-10).

Phase 5 objectives

The existing HHA method for vibration is based on ISO 2631
(1985), which uses an rms measure of vibration amplitude at the
seat. This standard was not intended for the evaluation of
repeated shocks. There is, therefore, a requirement for an
improved HHA method for repeated mechanical shock exposures in
TGVs which incorporates objective measures of human response
to acceleration and a clearly defined dose-effect relationship
between the vehicle acceleration history and injury.

The objective of Phase 5 was to develop a HHA model which
is suitable for evaluating exposures of soldiers to repeated
shocks in TGVs. The HHA model must be capable of predicting
injury risk to the operator or crew of a TGV given its seat
acceleration signature. The output of the HHA model must also be
compatible with the Risk Assessment procedure described in the US
Army HHA program AR 40-10.

98

Key features of the method for health hazard assessment

Exposure limits or guidelines must be related to a prediction of severe discomfort or probability of injury. Several key features which were identified in the Phase 4 experiments were included in the development of the HHA method. These included the non-linearity of both the acceleration transmission through the body and the subjective response to shocks.

The results of Phase 4 showed a distinct magnitude effect on the transmission ratios of accelerations from the seat to the spine. In both the positive and negative z axis, there were significant secondary impacts containing high frequency acceleration components after the initial shock. These effects are characteristic of a non-linear system and therefore cannot be represented by a simple linear model such as the DRI. The Phase 4 data also provided extensive new information on the shock magnitudes and frequencies most likely to cause severe discomfort, the exposure durations necessary to cause post-exposure stiffness or pain and the exposure required to reach the limit of a soldier's tolerance. Phase 4 experiments provided data and insight with which to approach both of these modeling issues.

The HHA method for mechanical shocks incorporates a number of features not provided in existing assessment models. The complete HHA method includes test conditions, types of measurements, data reduction and analysis techniques, as well as the predictive models necessary to translate measurements into a prediction of health risk.

The predictive models that comprise the HHA method include:

- biodynamic response models that predict spinal acceleration in response to acceleration input at the seat;

- regression models that predict peak compressive force at the L4/L5 lumbar joint, given peak spinal acceleration;

- a fatigue based dose model to quantify the cumulative effect of repeated mechanical shocks; and

99

- an injury probability model which relates the cumulative dose to the probability of spinal injury within a normally distributed population.

The output of the HHA method can be used to determine the appropriate RAC, as defined in the U.S. Army HHA Program (AR 40-10). A software version of the HHA method, complete with a graphical user interface (GUI), was developed to run under MATLAB™ software. A detailed description of the models and their development is included in the Phase 5 report.

The HHA method identifies both acute and chronic health risks resulting from either a few large amplitude shocks, or from prolonged exposure to vibration and repeated shocks due to travel over rough terrain. Within this context, the HHA method has applications outside of the military environment, in particular for assessment of the exposures encountered by vehicle operators in mining, forestry and construction activities.

Human response models

Biodynamic models have varied from relatively simple mass spring models containing one degree of freedom (Payne, 1992) to highly complex representations of the human body containing multiple degrees of freedom (Orne and Lui, 1971; Hopkins, 1972) and capable of simulating 3-dimensional motion (Belytschko and Previtzer, 1978; Amirouche and Ider, 1988).

A spinal model that has gained popularity is the DRI developed by Payne (1965, revised 1992) to predict the effect of vertical acceleration. The DRI is obtained from the response of a single mass-spring-damper system and is related to the peak compressive force developed in the spring and damper. A similar model was proposed by Fairley and Griffin (1989), based on data from humans exposed to lower amplitude vibrations. The Fairley-Griffin model contains a lower natural frequency ($fn = 5$ Hz) and higher critical damping ratio ($c = 0.48$) than the DRI ($fn = 11.9$ Hz and $c = 0.35$). Payne (1984) proposed similar models for the x axis ($fn = 10$; $c = 0.15$) and y axis ($fn = 7.2$; $c = 0.15$).

A more physiological approach is reported by Blüthner, Hinz and Seidel, (1986) and Seidel, Blüthner and Hinz (1986). The authors describe a biomechanical model in which the

compressive load at the L3/L4 disc is calculated from measures of trunk acceleration, upper torso mass and EMG activity. At 3 m·s^{-2}, and frequencies of 1 to 7 Hz, disc compressive forces ranged from 2 to 4.5 kN. The authors concluded that compressive force does not correlate uniformly with vibration intensity.

A number of limitations are apparent in existing human response models. Most models are based on a limited range of experimental data and have not been validated with repeated mechanical shocks. Few models include prediction of chronic health effects or tissue damage. Many of the models, including some of the more complex mathematical models, are restricted to uni-axial acceleration. Despite these criticisms, there have been several important contributions to biodynamic modeling which have direct relevance to the development of a HHA method.

Existing standards

A number of standards have been published for evaluation of WBV and shock. The most commonly used is the ISO 2631 (1974, 1985). It uses frequency weighting filters and the rms acceleration level to evaluate the health effects of exposure. The ISO 2631 is restricted to vibrations having a maximum crest factor of 6, and thus excludes exposures containing shocks. Nevertheless, this standard has been used in most reports of vehicle acceleration data.

The BS 6841 (1987) recommends that exposures containing shocks should be evaluated using a VDV of the form:

$$VDV = \left(\int_0^T a(t)^4 dt \right)^{\frac{1}{4}} \tag{10}$$

where a(t) represents the frequency weighted seat acceleration. The VDV does not provide limits for health effects, but a VDV of 15 is said to cause severe discomfort and to be approximately equivalent to the health exposure limit of the ISO 2631. A revision of ISO 2631 (1997) includes methods of evaluating repeated shocks, including the VDV, but exposure limits and risk of injury are not clearly defined.

An alternative method of evaluating repeated shocks is reported by the Air Standards Coordinating Committee (ASCC, 1982). It is based on the work of Allen (1977) and Payne (1978) who further developed the DRI model to account for the

101

health effects of multiple impacts. The ASCC uses a fatigue-failure model in which the number of shocks required to cause failure is a function of the predicted stress level (or DRI) and the estimated static failure stress (DRI_0). The health hazard guideline is based on a small amount of data (mainly spinal injury data from air crew ejection) at relatively high DRI values and it is limited to positive z axis shocks. Payne, Brinkley, and Sandover, (1994) have shown that the DRI provides a good correlation with subjective perception of shocks. However, Anton (1986) examined 223 air crew ejections and found the DRI to be a poor predictor of injury. Despite these limitations, the DRI is the only model in which the output is related to injury data, and for which some validation data have been reported. Thus the ASCC offers the best available guide to the HHA of repeated shocks at the present time.

Only the ASCC standard provides a prediction of severe discomfort and percent injury level. However, these limits are based on the output of a model which has been shown to be a poor predictor of spinal transmission (Phase 4 report: Cameron et al. , 1996). In addition, the suggested dose level for severe discomfort contained in the BS 6841 (i.e., VDV less than or equal to 15) does not appear to be applicable to a military population. The Phase 4 study proved that most soldiers are capable of tolerating a daily VDV of approximately 60. It should be noted that at this level of exposure, some subjects experienced a high level of discomfort and residual stiffness post exposure. However, due to the non-linear nature of the VDV model, a VDV of 60 represents over 250 times the number of shocks required to attain a VDV of 15.

Development of the health hazard assessment method

Any method of HHA capable of predicting the risk of injury from repeated mechanical shocks must be based on data from a wide range of studies encompassing human response, injury incidence, material properties and theoretical models. Therefore, the approach adopted in this study was to construct a HHA method which is based on components selected from existing models, human response data, tissue characteristics and injury data. From a review of the literature, it was established that there was insufficient experimental data on human response to repeated shocks to either test existing models or develop a new HHA method. Thus, during Phase 4, a number of experiments were

102

performed in which volunteers were exposed to a range of shock profiles and prolonged repeated shock exposures typical of those measured in TGVs (Cameron et al., 1996).

Although there was no objective evidence of injury to organ systems or tissues in the biochemical measures, and little evidence of muscle fatigue after exposure to severe motion in either Phase 3 or Phase 4, there was consistent subjective feedback regarding degraded physical status and perception of motion severity, fatigue, and discomfort. It was clearly demonstrated that the motion conditions in these experiments could result in extreme soreness and pain. Given the lack of objective evidence of injury and the relatively low levels of muscle activity indicated by EMG, it is likely that this soreness was related to inflammation or damage to spinal structures (i.e., vertebrae, intervertebral discs, ligaments). Furthermore, long-term exposure to vehicle motion has been associated with degenerative changes and injury to these structures (e.g., Boshuizen, Bongers and Hulshof, 1990; Wikström, Kjellberg, and Landström, 1994). Hence, an estimate of stress in the spine, combined with known material properties of vertebrae and discs (e.g., Brinckmann, 1988; Hutton and Adams, 1987; and Porter, Adams, and Hutton, 1989) may provide a good estimation of the probability of acute injury. Incorporation of existing models of mechanical fatigue of tissue (Lafferty, 1978; Sandover, 1983, 1986; Hansson, Keller, and Spengler, 1987) will extend the HHA model to the case of cumulative damage or degeneration over the longer term.

Ideally, the structure of the new HHA should include the following features:

- a means of predicting human spinal response (acceleration) in 3 axes;

- a means of relating biodynamic response (acceleration) to force (or tissue stress) within the body;

- a biomechanical model capable of computing internal forces in response to shocks;

- the ultimate (compressive) strength of the L4/L5 vertebral joint and the fatigue characteristics in response to cyclic loading;

- a means of predicting the cumulative effect of repeated mechanical shocks (a fatigue based dose model); and

- a means of assessing probability of injury, based on the population variance in the data related to maximum strength of vertebrae and/or acute injury.

A review of existing models and standards established that none satisfies all of the above criteria. From the above requirements, a new HHA structure was developed which integrates information from four distinct models:

- dynamic response models which predict peak accelerations of the lumbar vertebrae in the x, y and z axes in response to mechanical shocks input at the seat;

- a biomechanical model which analyzes spinal compression in response to shocks input at the seat;

- a dose model for exposure to repeated shocks based on prediction of spinal compressive forces and material fatigue failure theory; and

- an injury risk model based on the strength of vertebrae, population variance and the probability of failure.

Dynamic response models of acceleration in the x, y and z axes

As a first step, the characteristics of existing dynamic response models and filters were compared with the spinal acceleration data collected in Phase 4 (Cameron et al., 1996). Seated subjects were exposed to a series of shocks input at the seat in the x, y and z axes. Each shock was in the form of a single damped sinusoid, having a fundamental frequency between 2 to 20 Hz and amplitude of ± 0.5 g to ± 4 g. Acceleration was measured at the seat and at the skin surface over the lumbar vertebrae. Subjects were also asked to rate the subjective severity of each shock. It was found that the BS 6841 filter and DRI z axis model underestimated spinal transmission of the larger amplitude (2, 3 and 4 g) shocks in the z axis, and did not accurately reproduce the spinal response to negative z axis shock input at the seat. In addition, the spinal response in this axis was non-linear with amplitude. Although the response to seat accelerations in the x and y axes was found to be approximately linear, the DRI x and y axis models greatly overestimated transmission in these axes (Cameron et al., 1996).

104

Based on these findings, a series of dynamic response models were developed and tested using system identification techniques and the experimental data collected in this study. The models developed provide a continuous prediction of the x, y and z axis acceleration of the lumbar spine in response to the x, y and z axis shock input at the seat.

In the x and y axes, two separate strategies were investigated. The x and y axis responses were first modeled in the form of linear difference equations, using MATLAB™ systems identification software. The responses were also modeled using a mechanical analog, similar to that of the DRI, consisting of a mass, spring and damper. The natural frequency, fn, and critical damping ratio, c, of the models were adjusted to provide a best fit between the experimental data and the model output. Although the linear difference equations gave slightly better results in terms of both rms and rmd error, the analog models provided a more stable frequency response over the range of testing (0 to 80 Hz). Hence, in the HHA method the spinal response to acceleration in the x and y axes was modeled using a common second order linear model having the parameters fn = 2.125 and c = 0.22 (Morrison et al.,1997).

As the z axis response was found to be non-linear (Cameron et al., 1996), a different strategy was used to model the response in this axis. A recurrent neural network (RNN) was developed and trained to represent the system dynamics using the acceleration data measured in Phase 4. The RNN predicts acceleration output at the lumbar spine based on the previous acceleration inputs at the seat and previous predicted outputs. A series of neural networks were trained using samples of input-output data selected from a typical subject and then tested using unseen data. The best RNN developed consisted of 12 input processing elements (PEs), 7 hidden layer PEs and 1 output PE (Figure 46). Although more difficult to train, the RNN proved to be more desirable than a linear difference equation because it predicted the spinal response more accurately over the range of shock amplitudes tested (negative 2 g to positive 4 g). The quality of the output wave forms achieved using the RNN were superior to those predicted by the DRI or the BS 6841 frequency weighting filters. Details of the RNN development are provided in the Phase 5 report (Morrison et al., 1997).

Biomechanical model

A biomechanical model was developed which calculates the compressive force at the L4/L5 lumbar joint. The model utilizes measured human response data as input. Thus, it is not a predictive model and a biomechanical analysis using data measured from the vehicle operator does not form part of the HHA method. The purpose of the biomechanical model, together with the experimental data obtained from Phase 4, was to calculate the levels of compressive force generated at the L4/L5 vertebral joint in response to mechanical shocks in the x, y and z axes. The peak spinal accelerations predicted by the dynamic response models (above) were then related to the compressive force acting on the L4/L5 vertebral joint by means of a series of reegression equations.

Input to the biomechanical model consisted of position and acceleration data of the upper body in the x, y and z axes, and abdominal pressure. The data were collected in Phase 4. Abdominal pressure was measured by a specially designed rectal probe housing a miniature pressure transducer. Displacement was measured by infrared emitting diodes placed over the cervical (C7), thoracic (T4, T6, T8, T9, T10, and T12) and lumbar (L1 and L5) vertebra using an Optotrak system. The Optotrak data were used to determine the position and acceleration of the upper body center of mass.

The initial biomechanical model considered the upper body mass as a rigid body as in the model of Seidel, Blüthner and Hinz (1986). Results obtained proved this model to be impractical for large amplitude (i.e., greater than 1 g) shocks. The mass of the upper torso was therefore partitioned into two compartments representing the spine and soft tissues (Figure 47). The two compartments were each further subdivided into three spinal levels in order to accommodate the effects of spinal flexion on acceleration forces. The model computes forces and moments due to the linear and angular acceleration of the upper body mass and due to intra-abdominal pressure. Abdominal and spinal muscle forces and lumbar compressive force at the L4/L5 joint are then determined using an anatomical model and the principle of dynamic equilibrium.

The human response data collected during Phase 4 were used as inputs to the model, with output being the compressive (Cz) and shear (Cx, Cy) forces at the L4/L5 vertebral joint. Peak

compressive forces were calculated for a series of shocks ranging from negative 4 g to positive 4 g in the x axis, positive 0.5 to positive 4 g in the y axis and negative 2 g to positive 4 g in the z axis. Compressive and shear forces resulting from z axis shocks were larger than those for x or y axis shocks. These estimates of joint force, together with cadaver data of the ultimate strength of vertebrae, were subsequently used in a repetitive stress dose model in order to establish risk of injury.

Repetitive stress dose model

The structure of a dose model was developed which incorporates both a theory of fatigue failure and the material properties of the human L4/L5 vertebral joint. The dose model is based on the fatigue theory of Miner (1945) and the proposals of Payne (1976), Allen (1977) and Sandover (1983) that damage to the vertebrae due to repetitive shocks can be predicted using the concept of fatigue failure.

Miner (1945) proposed that the degree of fatigue (D) of a material subject to repeated stress can be expressed by the ratio (n_i/N_i), where:

n_i = the number of cycles completed at stress S_i; and

N_i = the number of cycles required to cause failure.

Thus, failure occurs when D = 1. This relationship can be generalized to any number of stress levels and cycles and expressed as in the form:

$$D = \sum (n_i/N_i) \tag{11}$$

In addition, the experimental results of Lafferty (1978) and Carter et al. (1981) show that when bone is repeatedly stressed, the number of cycles (N_i) required to cause failure can be modeled as:

$$N_i = (S_u/S_i)^x \tag{12}$$

where S_u = static failure stress, and S_i = applied repetitive stress level, and x is a constant for each material (Sandover, 1986).

107

The equivalent static stress level, S_e, which will produce a fatigue of D from a single loading can be written as:

$$S_e = \{\sum[n_i(S_i)^x]\}^{1/x} = S_u \cdot D^{1/x} \tag{13}$$

The equivalent stress value, S_e, can be considered as the stress "dose" applied to the material. This relationship is of a similar form to the existing DRI dose function (Payne, 1991).

By substituting Cz_i (the compressive force obtained from the biomechanical model) for S_i (stress) in the above equation, a spinal compression "dose value" (Cz_e) was obtained for the lumbar vertebrae in which Cz_i represents the peak lumbar compressive force due to shock i, and Cz_u represents the ultimate compressive strength of the lumbar L4/L5 joint (i.e., the compressive force required to cause injury). An exponent of x = 6 was chosen for the dose model based on the available literature for fatigue failure of bone (Lafferty, 1978; Carter et al., 1981; Brinckmann, Biggeman, and Hillweg, 1989). The ultimate strength of the L4/L5 spinal unit Cz_u was defined as 10,093 N, based on the combined experimental data of (Hutton and Adams, 1982 and Porter, Adams and Hutton, 1989).

Integration of the biodynamic and biomechanical models with the repetitive stress dose function

In order to develop an HHA method that could be related to existing knowledge of tissue properties, a biomechanical model was applied to estimate internal vertebral loading in response to individual shocks. The biomechanical model offers the advantage of a detailed analysis of lumbar compressive forces. However, it is an inverse dynamic model that requires displacement and acceleration data measured from the soldier as input. Hence, it is not a predictive model in the same manner as the dynamic response models. In these models the human acceleration response is predicted directly from the acceleration data input at the seat (i.e., the vehicle shock signature). Therefore, the biomechanical model was implemented in conjunction with the dynamic response models to determine the peak compressive force generated at the L4/L5 joint in response to shocks measured from a vehicle.

The output of the biomechanical model obtained from the Phase 4 data was compared with the outputs of the dynamic response models developed for the HHA. Regression functions were computed which related the peak compressive force developed at the L4/L5 joint to the corresponding spinal acceleration in the x, y or z axis. A separate regression equation was computed to relate the compressive force at the joint to the lumbar acceleration response to shocks in each axis (and for each direction in the x axis). With the aid of these relationships, the peak spinal accelerations predicted by the dynamic response models (in response to shocks input at the seat) can be used to estimate the corresponding peak lumbar compressive forces. These compressive force values are then inserted into the repetitive stress dose model above to obtain an accumulated compression dose measure in Newtons of force. Details of this process are described in the Phase 5 report (Morrison et al., 1997).

Injury risk model

The output of the dose model provides a single compression dose value for any given input (i.e., seat acceleration time series) to the dynamic response models. In accordance with normal biological variation, there will exist a range of dose values at which individual operators might be expected to experience injury or health effects. Hence, rather than associating the presence or absence of injury with a discrete dose value of 10,093 N (i.e., $Cz_e = Cz_u$) it is more practical to express the health effect of any dose in terms of the probability of sustaining injury. This can be achieved by relating the computed dose value to a cumulative probability function (Figure 48).

The probability of injury (or compression failure) is based on the distribution of a normal variable, which can be calculated using the relationship:

$$\Phi = f \ (\ Cz_e, \ Cz_u, \ \sigma \ ) \tag{14}$$

where: Φ = probability of injury due to material fatigue and σ = standard deviation of the ultimate strength Cz_u.

The variance (σ) of the underlying probability density function can be derived from cadaver data on the static strength of the vertebra-disc complex. The injury risk model was implemented using a mean compressive strength value of

109

Cz_u = 10,093 N and a standard deviation of σ = 1,926 N derived from Hutton and Adams (1982) and Porter, Adams and Hutton (1989). These data were considered to be the most reliable information available in terms of measurement technique and the age range (19 to 46 years) of cadaver specimens. Details of the injury risk model are provided in the Phase 5 report.

Incorporation of model components into a health hazard assessment method

The dynamic response models, biomechanical model output, the lumbar compression dose model and injury risk model were combined to produce the HHA method. The input consists of seat acceleration time series in the x, y and z axes. The method output is the probability of injury calculated for a specified exposure duration.

A fundamental requirement of the HHA method is that it must be integrated into the existing U.S. Army HHA Program (AR 40-10). Because of this requirement, the probability of injury predicted by the HHA method determines the hazard severity level on a scale of I to IV. The hazard severity level is combined with the probability of occurrence (determined by the vehicle type and its operating environment) and used to determine the appropriate risk assessment code (RAC) as defined in the AR 40-10 (1991).

A software version of the HHA method with a graphical user interface (GUI) was developed using MATLAB™ software. The HHA GUI selects the input data files of vehicle seat acceleration in the x, y and z axes, the intended exposure duration (days, hours, minutes, seconds), and the expected probability of occurrence of this exposure (ranked A to E according to whether the particular exposure is likely to be frequent, occasional or improbable). The program then calculates the spinal acceleration response, the compression dose value and injury probability. The resultant hazard severity level and RAC value are then reported on the HHA GUI (Figure 49). The HHA GUI also provides options to display the seat and spinal accelerations, the lumbar compression dose value and the probability of injury as a function of exposure time.

Application of the HHA method

The HHA method described above predicts the risk of injury and assigns the RAC for a specified test condition, exposure duration and hazard probability. This approach provides the risk of acute injury from a well defined single exposure condition that may last a few hours or a few days. Two other exposure scenarios may be of interest for the evaluation of health hazard: chronic injury from regular exposure to repeated shocks during a number of years; and complex mission profiles that may involve exposure to multiple acceleration conditions. The RAC can be determined for either of these scenarios if the appropriate exposure duration and probability of occurrence are defined.

The HHA method was tested using a selection of repeated shock profiles and exposure durations varying from 6 hours to 20 years. The input data for this simulation was obtained from experimental data collected in Phase 3 and Phase 4. Results indicated that the most severe exposure containing 2 and 4 g z axis shocks would cause marginal injury in one day, but could lead to severe injury if soldiers were exposed on a daily basis for a prolonged period. By comparison, exposure to rms vibration levels equivalent to the ISO 2631 health guidance limit provided negligible injury risk when accumulated over a period of 20 years. Details of the analysis are provided in the Phase 5 report (Morrison et al.,1997). In Figure 50 the probability of injury predicted by the HHA is shown for one exposure condition (a 6 hour exposure duration with z axis shocks, including 32 ±2 g shocks and two +4 g shocks per 5 min).

Military vehicle HHA test protocol

A test protocol was developed for evaluation of military vehicles using the HHA method. It is the intention of the vehicle test and evaluation procedure to apply a standard method for evaluating the health risk associated with the vibration and impact environment of any Army vehicle. Knowledge of the health risk associated with specific operating situations can be applied during vehicle design and acquisition, or for planning operations and exercises to minimize both chronic and acute injuries to the soldier.

111

The test protocol includes:

- operating conditions under which a vehicle is to be tested;

- types of measurements to be made;

- methods of data reduction and analysis; and

- assessment of health hazard using risk assessment codes.

Details of the military vehicle HHA test protocol are provided in the Phase 5 report (Morrison et al., 1997).

Phase 5 conclusions and recommendations

In Phase 5, a health hazard assessment (HHA) method for exposure to repeated mechanical shocks was developed. The method introduces several innovative concepts that include and a non-linear z axis acceleration response model, the use of a biomechanical model, and cadaver data. The biodynamic and biomechanical models were developed with the aid of experimental data obtained from soldiers exposed to repeated mechanical shocks in the range of 0.5 g to 4 g in the x, y and z axes. The HHA method includes a dose model based on peak compressive forces at the L4/L5 lumbar joint and material fatigue, and an injury risk model based on strength of the L4/L5 spinal unit and population variance. The HHA method has undergone a limited validation with existing experimental and epidemiological data, with acceptable results. The model needs to be more rigorously tested against chronic injury data due to long term exposures to WBV and repeated shocks.

There are a number of limitations to the HHA method which will affect the accuracy of the hazard assessment. The probability of injury is based on a small amount of data describing vertebral fracture in the spinal units of cadavers. It is designed to represent male soldiers within the age range of approximately 20-40 years. The model does not take account of either the ability of biological material to recover through the repair process, or the decline of vertebral strength with age. However, it is proposed that this structure forms the basis of a HHA method, within which sub-components and parameters may be

112

adjusted as the outputs are more rigorously tested against experimental and epidemiological data.

Challenges and limitations

Throughout this project the research team faced a number of logistical challenges and limitations which were inherent in the study design. The limitations included, but were not limited to the following:

- developing a model of health risk from a database related to acute exposure to motion environments;

- detecting a measurable health-related change in response to exposure to a relatively low dose of vibration and repeated impact;

- ethical concerns of exposing subjects to risk of injury;

- relating motion signatures for vehicles and environments in which they are driven directly to chronic health effects reported in epidemiological literature;

- having a limited number of subjects that could be tested within the scope of the project relative to the large sample population required to validate sensitivity contours (Oborne, 1983); and

- determining the frequency range of motion in TGVs and developing realistic motion exposure experiments.

In Phase 3 and Phase 4 the limitations faced in the acquisition and interpretation of human response data included the following:

- all subjects were male, aged 19 to 40;

- subjects represented a relatively fit, military population;

- a relatively small number of subjects were tested;

- large inter- and intra-subject variability was observed;

- due to limitations in the excursions capability of the MARS, the experimental design included a limited range of motion environments with respect to shock amplitude (0.5 g to 4 g) or frequency (2 Hz to 20 Hz); and

114

- long duration experiments were limited to 7 hour
  exposures in one day, or 4 hours per day for
  5 consecutive days, which does not reflect a
  typical military mission.

These limitations, coupled with a lack of published
epidemiological data on the effect of repeated shocks, restricted
the data which were available to build and test the models which
were incorporated into the HHA method.

The HHA method developed in Phase 5 included the following:

- biodynamic response models;

- a biomechanical model; and

- dose and injury models.

The major limitation in the development of the biodynamic
response models was the limited range of shock signatures to
which subjects were exposed. Therefore, the linear response
models applied to the x and y axis data should only be
extrapolated beyond these limits with caution. The nonlinear
nature of the z axis model prevents its extrapolation beyond
the data used to train the recurrent neural network.

The biomechanical model was limited both by a lack of
published data on spinal shear, and the inability to directly
confirm the internal forces that were predicted as a result
of exposure to shocks.

The dose and injury models, which were based on fatigue of
the spinal unit, did not include the effect of either biological
recovery or the aging process. Therefore, this limits their
application to multi-exposure risk accumulation, particularly
over a number of years.

The proposed HHA method is based on theoretical and
experimental knowledge, but requires validation in the field
during typical military operations. Field validation would
include the incidence and characteristics of severe discomfort
and injury, the associated motion environment, and the
population demographics.

The accuracy of the HHA method presented herein is limited,
therefore, by the current knowledge of the human response to
repeated mechanical shocks.

115

Appendix A

The project team

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Research Engineer, B.C. Research Inc.

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Research Engineer, B.C. Research Inc.

   Other B.C. Research personnel who worked on this project
included: Dale Brown (Phase 2 and 3); Mark Garzone (Phase 4);
Gillian Gibbs (Phase 4); Steve Martin (Phase 4); Brian Remedios
(Phase 2 and 3); Laurel Ritmiller (Phase 4); Julia Rylands
(Phase 1 and 3); Judy Village (Phase 1,2 and 3); and Alan
Vukusic (Phase 4).

Appendix B

The project team biographies

Dr. Anthony Brammer, Ph.D. Physics (University of Exeter, 1967)

Dr. Brammer completed his undergraduate education and Ph.D.
in Physics at the University of Exeter, England before completing
a post-doctoral research fellowship in the division of Physics at
the National Research Council of Canada. He has been a sessional
lecturer in Physics at Carleton University, an adjunct professor
in the Department of Mechanical Engineering at the University of
Windsor, a lecturer for the National Research Council, and a
graduate supervisor for the Department of Mechanical and
Aeronautical Engineering at Carleton University. Presently he is
the Senior Research Officer for the Institute for Microstructural
Sciences, and has been the Senior Research Officer for the
Division of Physics at the National Research Council of Canada.
His research expertise includes: acoustic techniques and
instrumentation, hand-arm vibration, biodynamics, machinery
vibration, machinery noise, and noise exposure. Dr. Brammer has
been a visiting scientist at the Institute of Occupational Health
in Helsinki, Finland, the Department of Public Health at Kanazawa
University in Japan, and at the Department of Health Care and
Epidemiology at the University of British Columbia in Vancouver,
Canada. He has extensive peer reviewed publications.
Dr. Brammer has also received many awards and distinctions
for his contributions in the area of vibration and acoustics.
He is the Appointed Convenor in the current development of
ISO standards for vibration. He is a member of numerous
professional, learned, scientific, engineering, and technical
societies as well as Canadian and international committees.

117

<u>Dr. Barbara Cameron, Ph.D. Kinesiology (Simon Fraser University, 1992)</u>

Dr. Cameron is Director of the Ergonomics and Human Factors Group at B.C. Research Inc. Prior to this appointment, she worked for 2 years as a consultant to the company. She has extensive experience in environmental ergonomics, pre-employment testing for job selection, and the physiological and biochemical characteristics of fatigue. In addition to her research expertise, Dr. Cameron has developed and delivered full education packages for university courses and for adult education workshops. At the University of Calgary, worked as a research assistant studying thermoregulation in humans and animals. While completing her Ph.D. dissertation in work physiology at Simon Fraser University, she coordinated the Institute for Human Performance. She was responsible for performance testing and evaluation, and helped to coordinate a major collaborative research project between the University of Washington and Simon Fraser University in the hypobaric facility at SFU. She also assisted in the organization and implementation of regular pre-employment testing for firefighters, which included both physical and psychomotor evaluation of up to 1200 applicants. In her role as project manager for this study, Dr. Cameron coordinated a team of ergonomists, computer programmers, engineers, and consultants. She was responsible for all aspects of budget, administration, and timely delivery of reports. Dr. Cameron is also the project manager for a number of studies supported by Transport Canada and the Department of National Defence. Her project experience includes: effects of vessel motion on target detection in marine search and rescue; effects of extended crewing periods in Arctic icebreaking operations; evaluation of military tentage systems; and the development of occupationally based hearing and vision standards. Dr. Cameron completed a course in Industrial Ergonomics at the Harvard School of Public Health. She is a member of the B.C. Association of Kinesiologists, the Human Factors Association of Canada, the Human Factors and Ergonomics Society (U.S.), and the Ergonomics Society (U.K.).

<u>Dr. James Morrison, Ph.D. Bioengineering (Strathclyde University, 1967)</u>

Dr. Morrison has a B.Sc. in mechanical engineering and a postdoctoral research fellowship from Massachusetts Institute of Technology in Cambridge. Dr. Morrison is a Professor in Kinesiology and Associated Professor in Engineering Science at Simon Fraser University. He is also President of his consulting company, Shearwater Human Engineering Ltd. His areas of research include biomechanics, computer aided design, ergonomics and environmental ergonomics. Biomechanics research is centered on the modeling of human locomotion and analysis of muscle, joint and skeletal forces. Recently he has been investigating load transmission across long bone fractures. Computer Aided Design work involves automated manufacture of lower limb prostheses through computer generation of limb shapes from anthropometric data. Ergonomics research includes the measurement and analysis of WBV in humans and their interpretation in terms of acute and chronic health effects. Environmental ergonomics research includes thermal regulation, pressure physiology and design of breathing apparatus. He is investigating the physiological consequences of hypoventilation, CO_2 retention, respiratory adaptation, the interaction of CO_2 and N_2 narcosis, and impaired performance directed towards defining the physiological requirements of underwater breathing apparatus and developing new concepts in apparatus design. Dr. Morrison's expertise in the area of human response to vibration and mechanical shock has been recognized by his appointment as a technical delegate to the Standards Council of Canada (SCC) and as a representative of SCC to the International Organization for Standardization (ISO) subcommittee on human response to vibration and mechanical shock (ISO SC4 TC108). Dr. Morrison has supervised 28 graduate students, is a member of three learned societies, is Subject Editor of Ergonomics Journal, and is the President of the B.C. Chapter of the Human Factors Association of Canada.

Jordan Nicol, M.Sc. Electrical Engineering (Simon Fraser University, 1996)

Mr. Nicol has a B.Sc. and M.Sc. in electrical engineering with a specialization in biomedical engineering. His Master's work was focused on modeling the dynamic response of the human spine to mechanical shock and vibration. This modeling involved the development of an artificial neural network and two linear difference models. The models developed by Mr. Nicol and his co-workers were also presented as a proposed annex to the International Organization of Standardization for inclusion in the ISO 2631. The annex presents the mathematical basis for a model to predict lumbar spine acceleration from measured seat acceleration, including shocks. Mr. Nicol's master's program was funded jointly by the B.C. Advanced Systems Institute, Simon Fraser University and B.C. Research Inc. During Mr. Nicol's work at Simon Fraser University he also contributed to the development of an alignment device for below knee amputees. Mr. Nicol programmed a microprocessor which controlled three motors used to power an adjustable alignment device required in the fitting of an amputee's prosthesis.

<u>Dan</u> <u>Robinson,</u> <u>M.Sc.</u> <u>Kinesiology</u> <u>(Simon</u> <u>Fraser</u> <u>University,</u> <u>1991)</u>

Dan Robinson completed a B.Sc. in biochemistry at the University of British Columbia and an M.Sc. in Kinesiology at Simon Fraser University. His training has focused on evaluating human response to challenging work environments, using a wide range of physiological, biochemical and psychophysiological techniques. Mr. Robinson's project experience includes the investigation of physiological and biochemical effects of altitude sickness and adaptation to altitude, the effects of extended work days on psychophysiological function and health in underground mine workers, and the influence of pesticide exposure on tree planters. In addition, he has experience with occupational task analyses, process flow, work organization and systematic layout planning. He has applied this knowledge to the design of control rooms, manufacturing facilities, office environments and libraries. His Master's thesis examined human response indices of low level exposure to organophosphate and carbamate pesticides while tree planting. Pesticide exposure indices included blood and tissue assay for cholinesterase isozyme inhibition, sensory and motor nerve conduction velocities, and physical symptom evaluation. While employed by B.C. Research Inc., Mr. Robinson is completing the requirements for a Ph.D. in Kinesiology. His dissertation examines the influence of spinal musculature and intra-abdominal pressure on the biodynamic response to mechanical shocks. This work is contributing to the development of a standard for the U.S. Army to evaluate health risk from exposure to repeated mechanical shocks. Mr. Robinson's experience in the evaluation of human response to vibration and impact has been recognized by his appointment as a technical delegate to the Standards Council of Canada (SCC) and as a representative of SCC to the International Organization for Standardization (ISO) subcommittee on human response to vibration and mechanical shock (ISO SC4 TC108). Mr. Robinson is a full member of the Human Factors Association of Canada and a student associate of the Human Factors and Ergonomics Society (U.S.).

George Roddan, B.Sc.(Hons) Mathematics and Physics (Simon Fraser University, 1977) P.Eng.

George Roddan has worked as Research Scientist at the Ocean Engineering Center (OEC) at B.C. Research Inc. since 1981. During this time he has participated as a technical expert in over 250 successfully completed projects. The projects have consisted mainly of scale model studies to evaluate the performance of various types of marine craft ranging from barges and fishing boats to the latest designs of planing yachts, fast catamarans, and advanced marine vehicles. Mr. Roddan possesses advanced skills in instrumentation and computerized data acquisition. Many of the hydrodynamic studies performed at the OEC require the use of specialized instrumentation such as force and torque gauges, pressure transducers, gyroscopes and wave probes. Data from these various sensors is acquired using a variety of customized data acquisition programs. Mr. Roddan has overseen the development and implementation of both hardware and software elements of the current OEC data acquisition system. Mr. Roddan also has extensive experience in computer programming and data analysis. His programming abilities have been widely used in the OEC to develop custom analysis programs as well as numerical control programs (e.g., interfacing the computer to the hydraulic systems of the towing tank wave maker). In addition, Mr. Roddan has experience in computerized spectral analysis of vibration and shock phenomena, and has been a key contributor to the current study for the U.S. Army. Mr. Roddan was involved in the analysis of vibration data gathered in laboratory simulations of the vehicle environment. He also generated the digital control files which were used to control a three-axis shaker table used to simulate the vehicle environment. The control signals were based on a protocol developed by the principal investigator, and approved by an ethics review committee.

Julie Springer, M.Sc. Mechanical Engineering (University of Calgary, 1994)

Julie Springer's educational background includes a B.Sc. (U. of Calgary, 1990) in mechanical engineering and a minor in computer integrated manufacturing as well as an M.Sc. (U. of Calgary, 1994) in mechanical engineering with a focus on biomechanics and prosthetic design. Ms. Springer combined her education in engineering with anatomy and anthropometry. At the University of Calgary and Clynch Technologies Inc., she designed and developed supporting structures for computer-aided prosthetic socket design systems. She continued working in this field for two years at Simon Fraser University with Dr. J. Morrison, and also began contacts in ergonomics at wood processing mills. Ms. Springer has gained extensive expertise in computer aided design using AutoCAD. She has also conducted usability testing on the fit and comfort of lower limb prosthesis and worked in development of tools to aid in adjustability of prosthetic alignment devices. Currently employed by B.C. Research Inc. in the Ergonomics and Human Factors Group, Ms. Springer is working on a project with the U.S. Army on the development of an exposure standard for repeated impact in tactical ground vehicles. She has also worked on various industrial ergonomics contracts including: vibration assessment and ergonomic evaluation of seating in locomotive cabs; assessment of hand-arm segmental vibration and whole body vibration in mining operations; development and delivery of ergonomics training to hospital design groups; ergonomic input at a soderberg aluminum smelter; ergonomic input in the laboratory design at a pulp and paper mill; ergonomic assessment of an MD6 compression tool; and ergonomic assessment of VDT workstations and laboratories at B.C.'s Women's and Children's Hospital. Ms. Springer is a full member of the International Society of Automotive Engineers (SAE), and is currently applying for her professional status in the Association of Professional Engineers, Geologists and Geophysicists of Alberta (APEGGA). Ms. Springer is also an associate member of the Human Factors Association of Canada (HFAC).
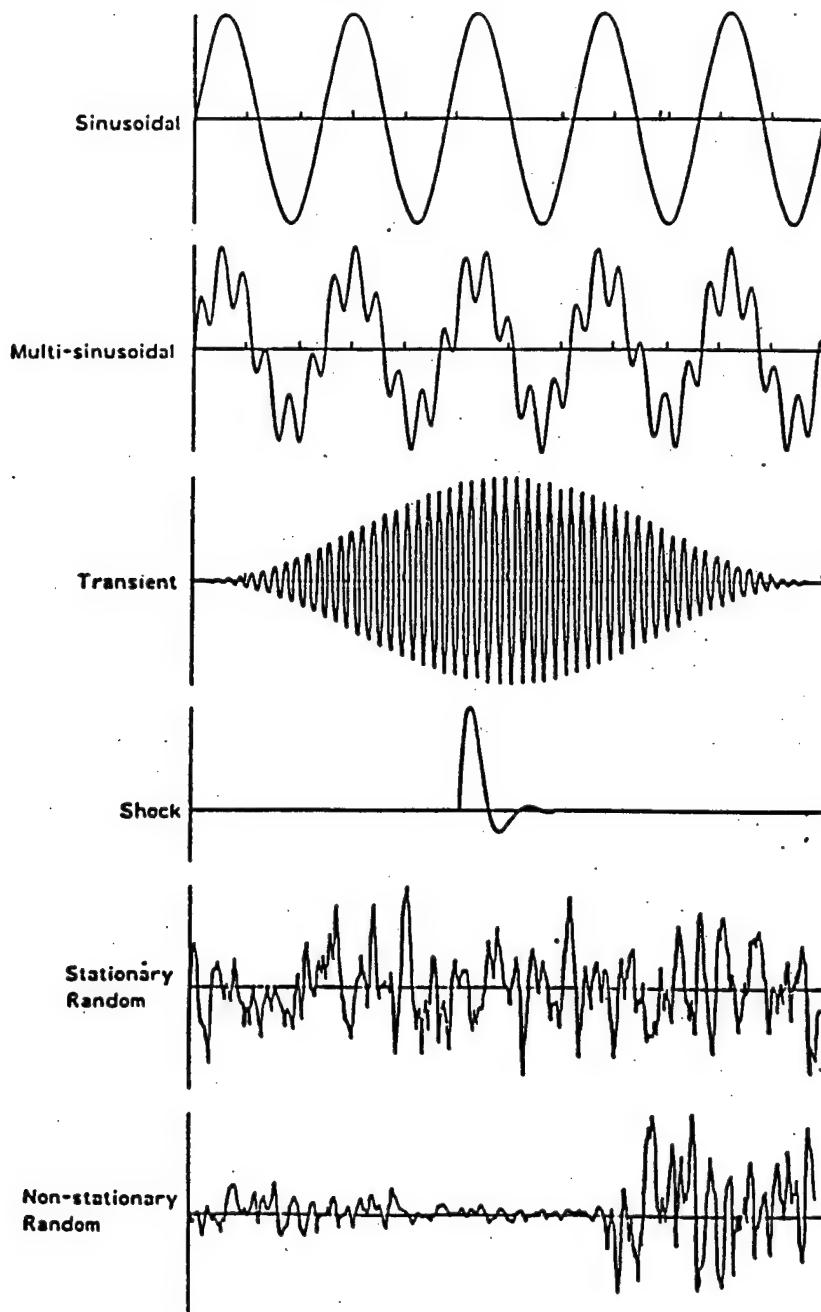
Figures

Figure 1. Examples of deterministic and random vibration waveforms, and shocks (after Griffin, 1990).
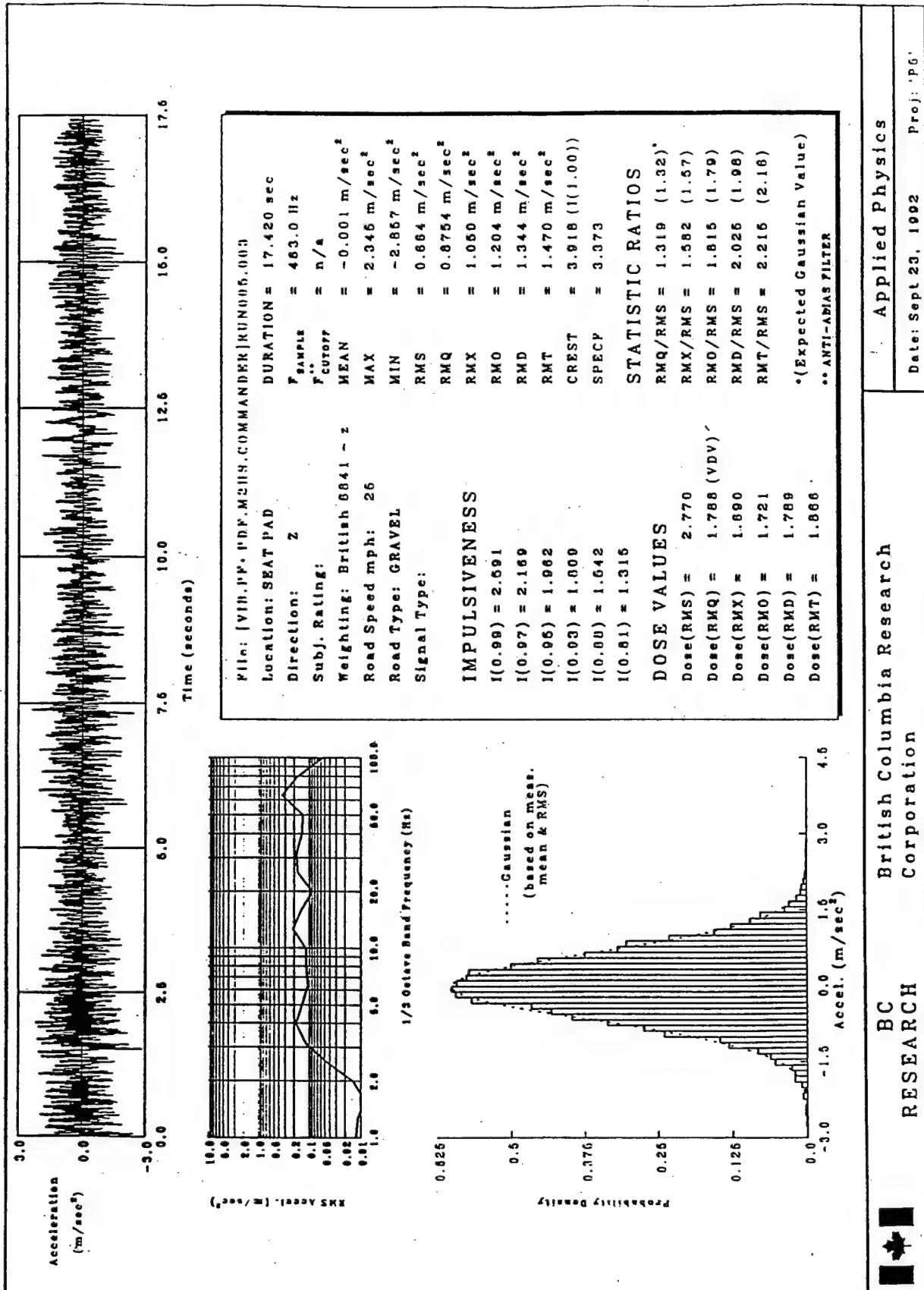
Figure 2. Seat motion analyzed using frequency weighting in British standard, BS 6841 (1987).
Signal type 1: Gaussian random motion

125
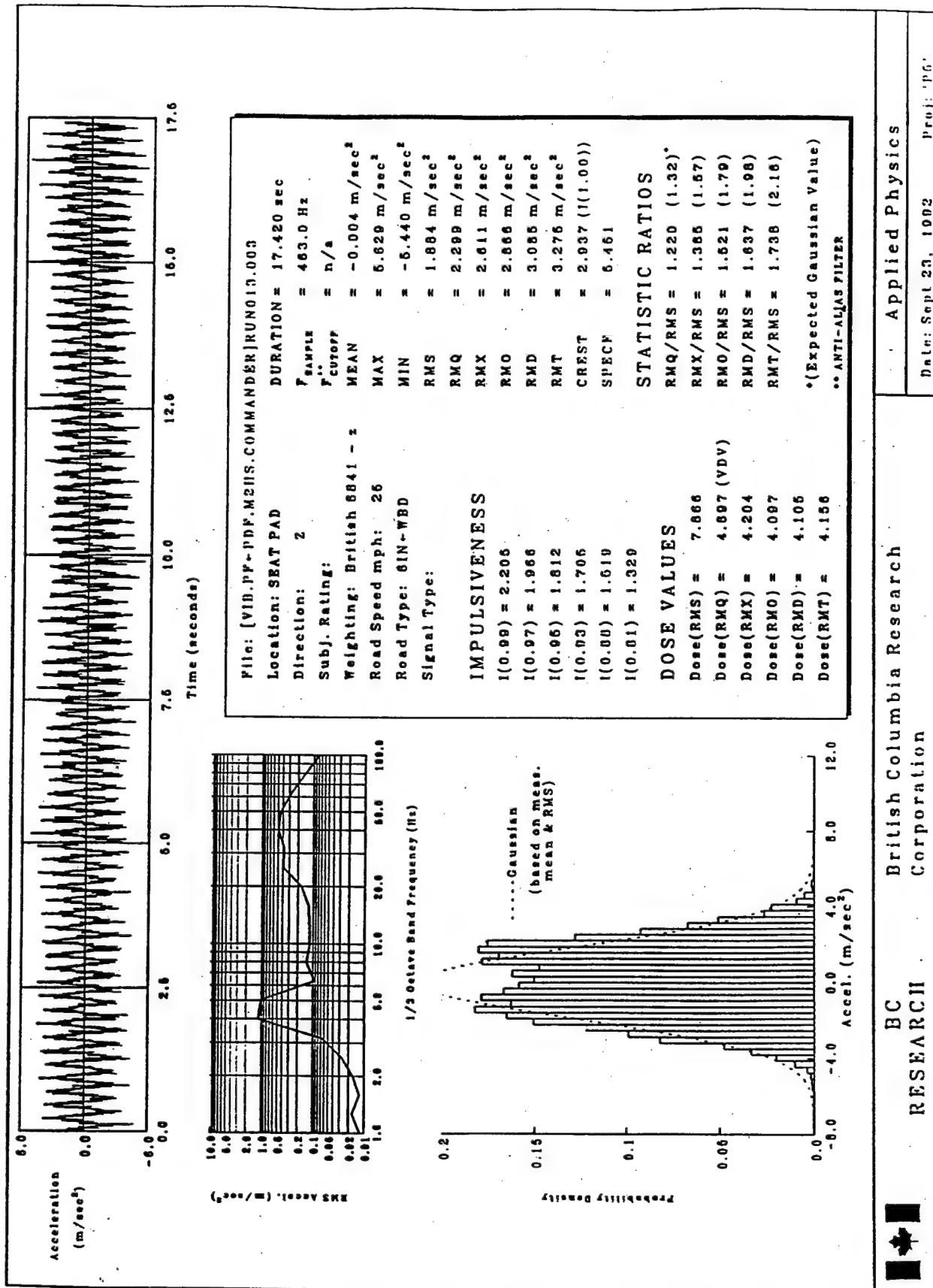
Figure 3. Seat motion analyzed using frequency weighting in British standard, BS 6841 (1987).
Signal type 2: Near sinusoidal high-frequency motion

File: [VIB.PF←PDF.M1-HTT.DRIVER]RUN026.002

Location: SEAT PAD	DURATION = 20.162 sec
Direction: Y	F_{SAMPLE} = 416.7 Hz
Subj. Rating:	F_{CUTOFF}^{**} = n/a
Weighting: ISO 2631 - x,y	MEAN = 0.002 m/sec²
Road Speed mph: 5	MAX = 3.132 m/sec²
Road Type: XC#4	MIN = -4.590 m/sec²
Signal Type:	RMS = 0.971 m/sec²
	RMQ = 1.589 m/sec⁴
IMPULSIVENESS	RMX = 2.058 m/sec⁶
	RMO = 2.407 m/sec⁸
I(0.99) = 3.530	RMD = 2.674 m/sec²
I(0.97) = 2.964	RMT = 2.864 m/sec²
I(0.95) = 2.395	CREST = 3.978 (I(1.00))
I(0.93) = 1.922	SPECF = 6.304
I(0.88) = 1.395	
I(0.81) = 1.003	STATISTIC RATIOS
	RMQ/RMS = 1.637 (1.32)*
DOSE VALUES	RMX/RMS = 2.121 (1.57)
	RMO/RMS = 2.480 (1.79)
Dose(RMS) = 4.358	RMD/RMS = 2.755 (1.98)
Dose(RMQ) = 3.388 (VDV)	RMT/RMS = 2.971 (2.18)
Dose(RMX) = 3.396	*(Expected Gaussian Value)
Dose(RMO) = 3.504	** ANTI-ALIAS FILTER
Dose(RMD) = 3.611	
Dose(RMT) = 3.704	

Acceleration (m/sec²)

Time (seconds)

RMS Accel. (m/sec²)

1/3 Octave Band Frequency (Hz)

Probability Density

Accel. (m/sec²)

..... Gaussian (based on meas. mean & RMS)

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Applied Physics

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Figure 4. Seat motion analyzed using frequency weighting in British standard, BS 6841 (1987). Signal type 3: Transient sinusoidal motion
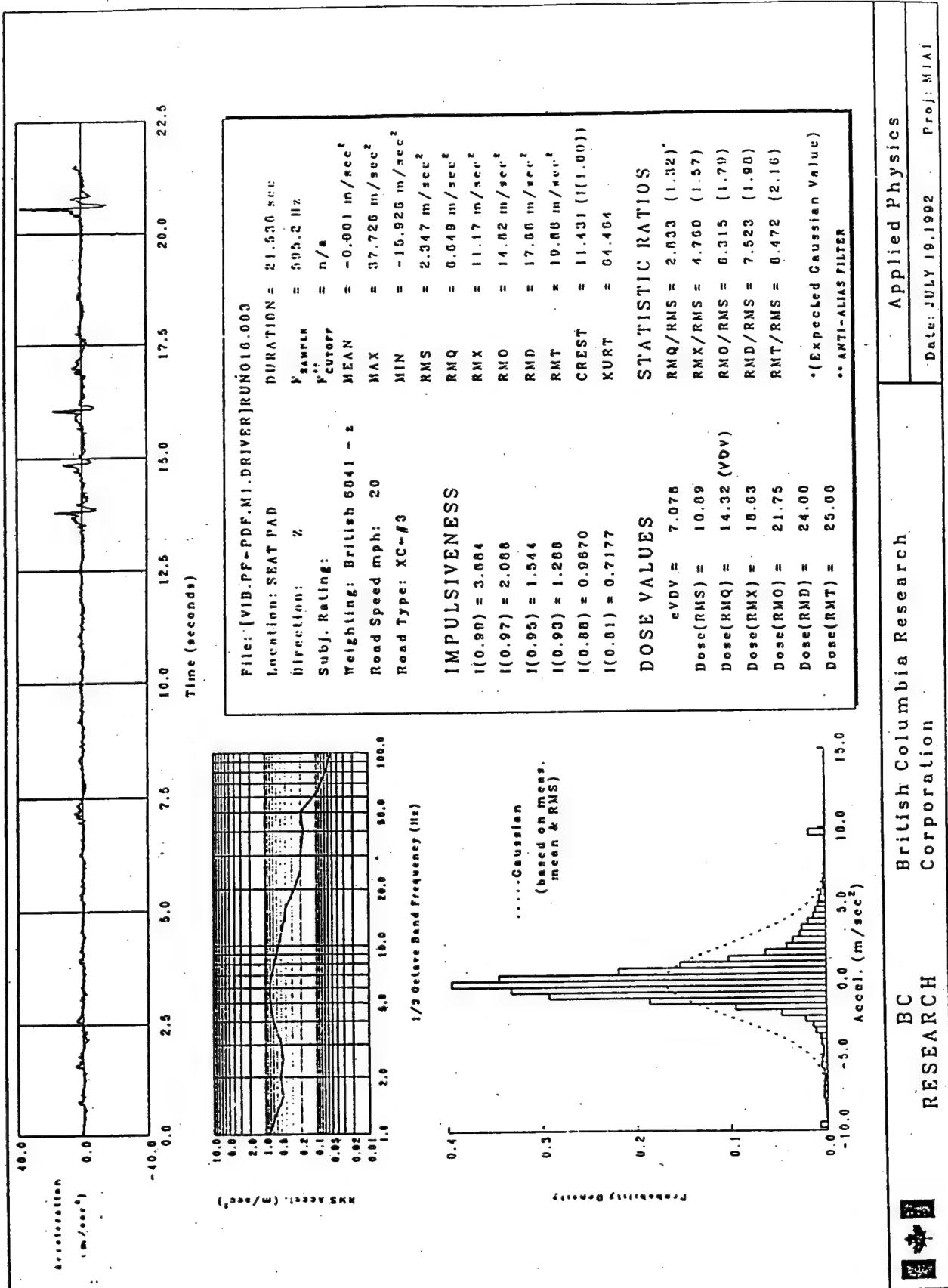
Figure 5. Seat motion analyzed using frequency weighting in
British standard, BS 6841 (1987).
Signal type 4: Impulses (shocks)
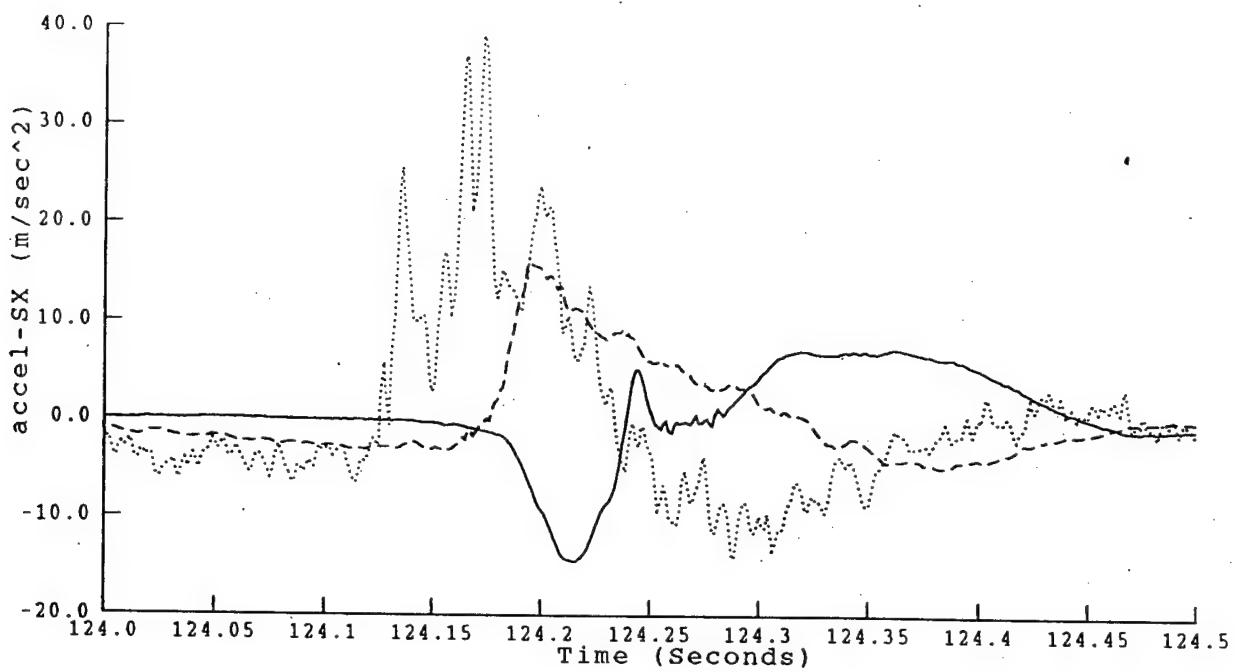
Figure 6.   Unfiltered acceleration measured at the seat, lumbar and thoracic spine for a 3 g, 4 Hz x axis shock.   *Dotted line: seat Sx; broken line: lumbar L2 x; full line: thoracic T1 x.*
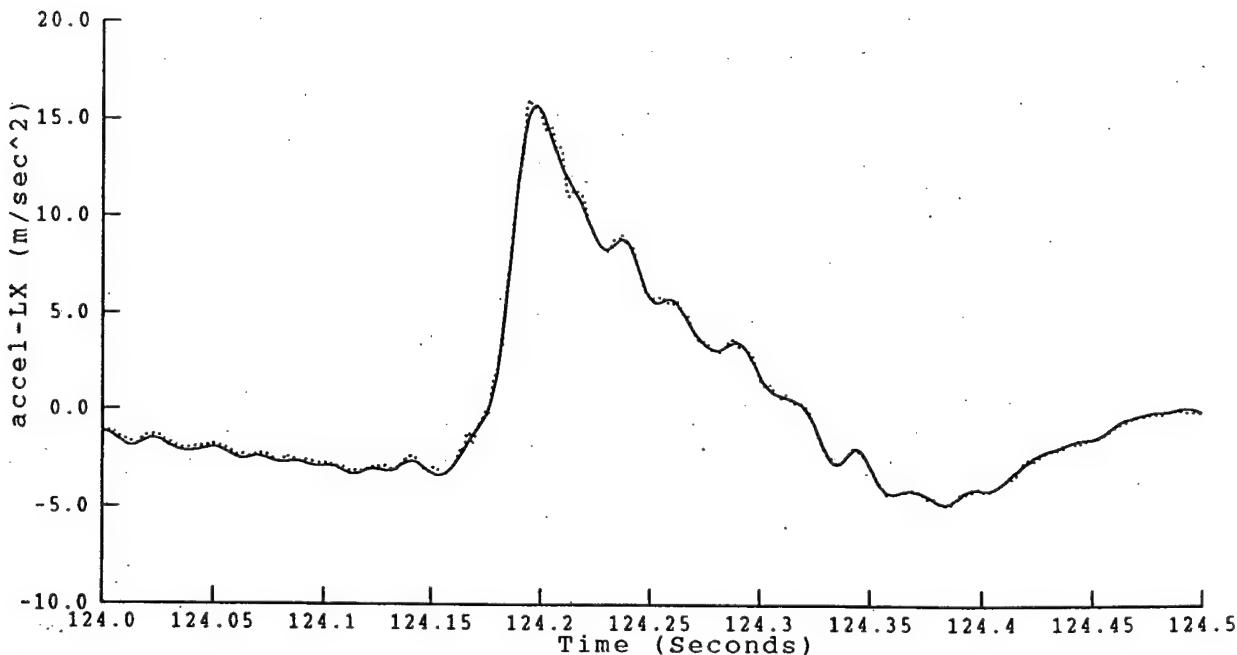

Figure 7.   Acceleration at the spine (L2 x) for a 3 g, 4 Hz x axis shock. *Dotted line: unfiltered data; full line: band pass filtered at 0.5 Hz to 60 Hz.*
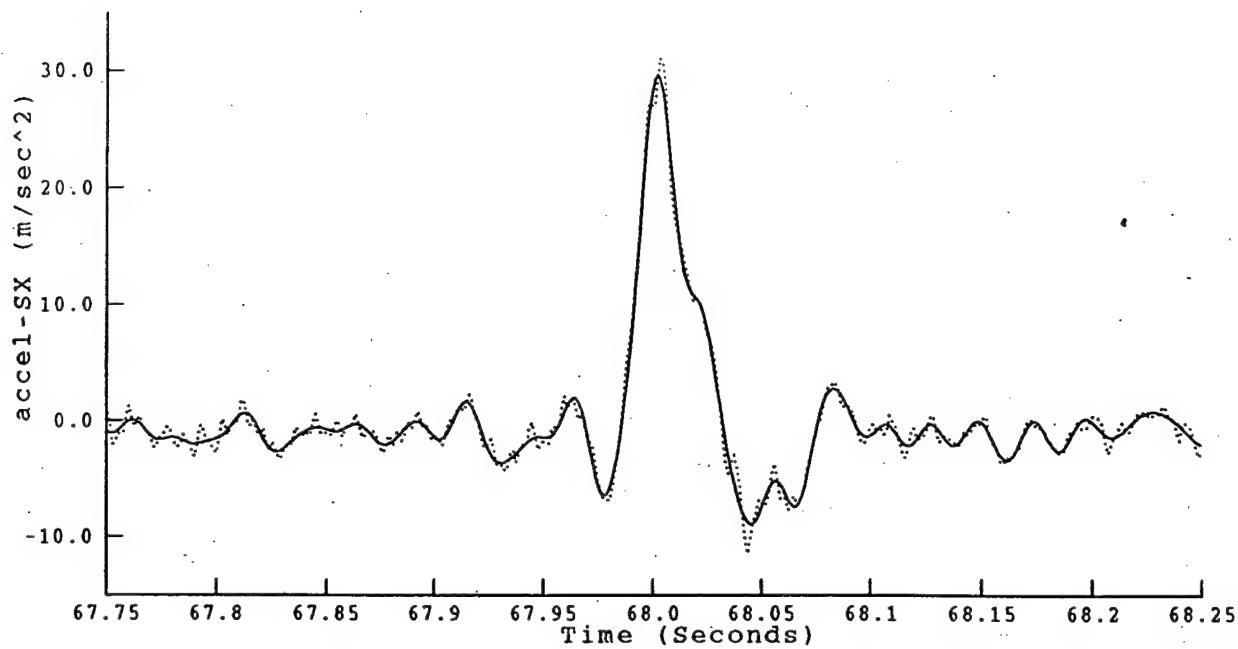
Figure 8. Acceleration at the seat (Sx) for a 3 g, 11 Hz x axis shock. *Dotted line: unfiltered data; full line: band pass filtered at 0.5 Hz to 60 Hz.*
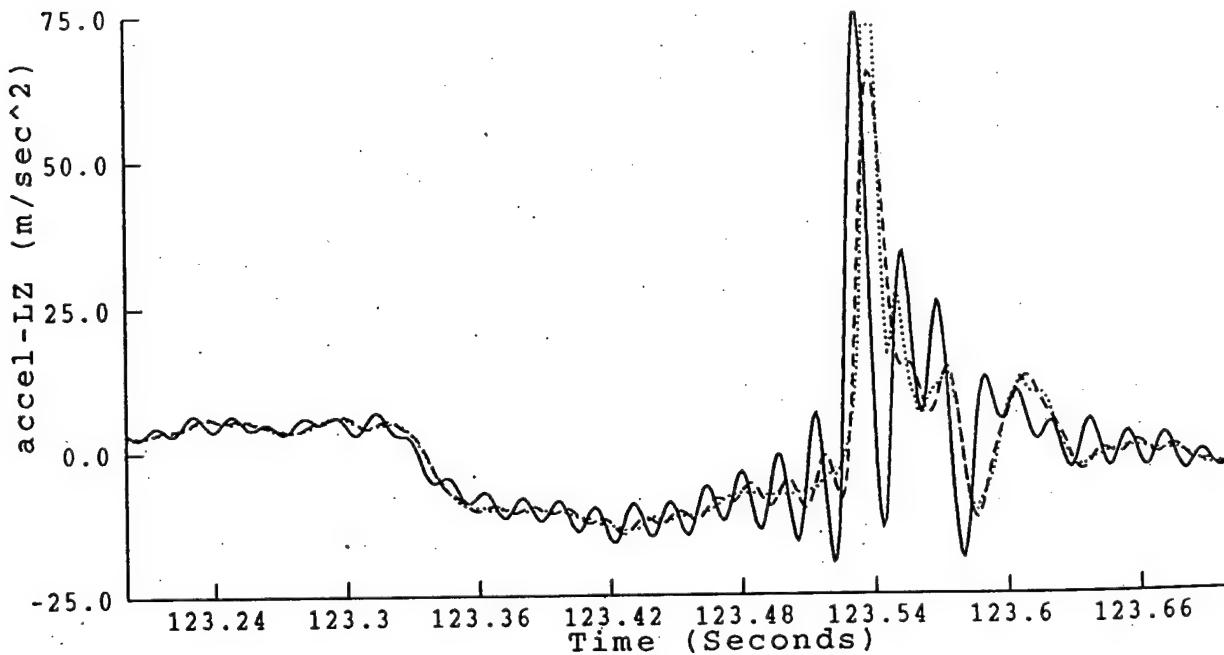
Figure 9. Acceleration at the spine (L4 z) for a -3 g, 4 Hz z axis shock. *Dotted line: unfiltered data; broken line: filtered data; full line: filtered data multiplied by inverse transfer function.*
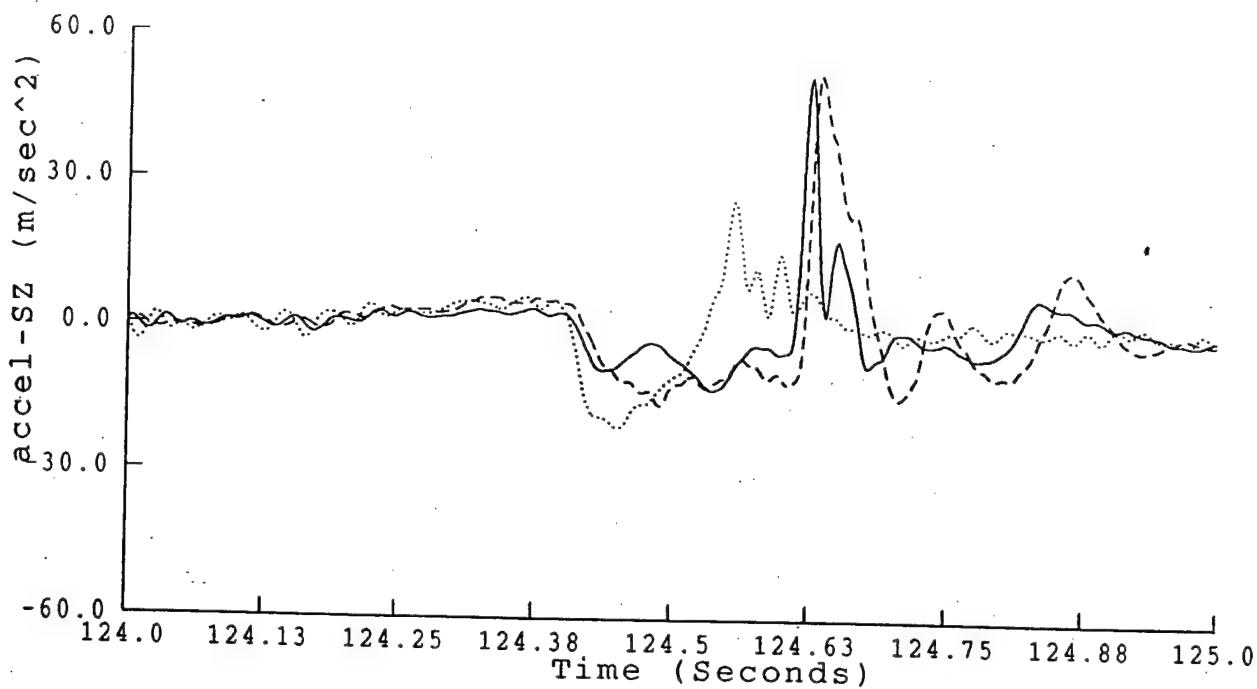
Figure 10. Acceleration measured at the seat, lumbar and thoracic spine for a -3 g, 4 Hz z axis shock. *Dotted line: seat Sz; broken line: lumbar L4 z; full line: thoracic T3 z.*
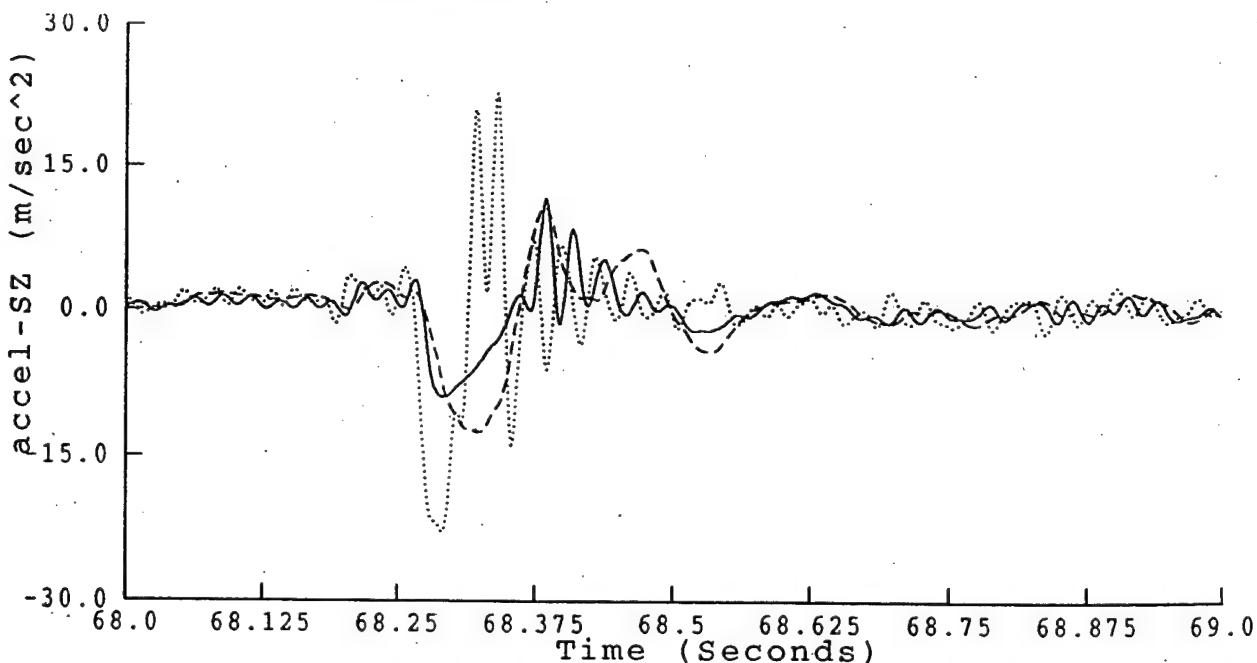
Figure 11. Acceleration measured at the seat, lumbar and thoracic spine for a -3 g, 11 Hz z axis shock. *Dotted line: seat Sz; broken line: lumbar L4 z; full line: thoracic T3 z.*

Figure 12: (a,b,c) Spine (T3) z acceleration response to seat z acceleration for 3, 2, & 1 g shocks
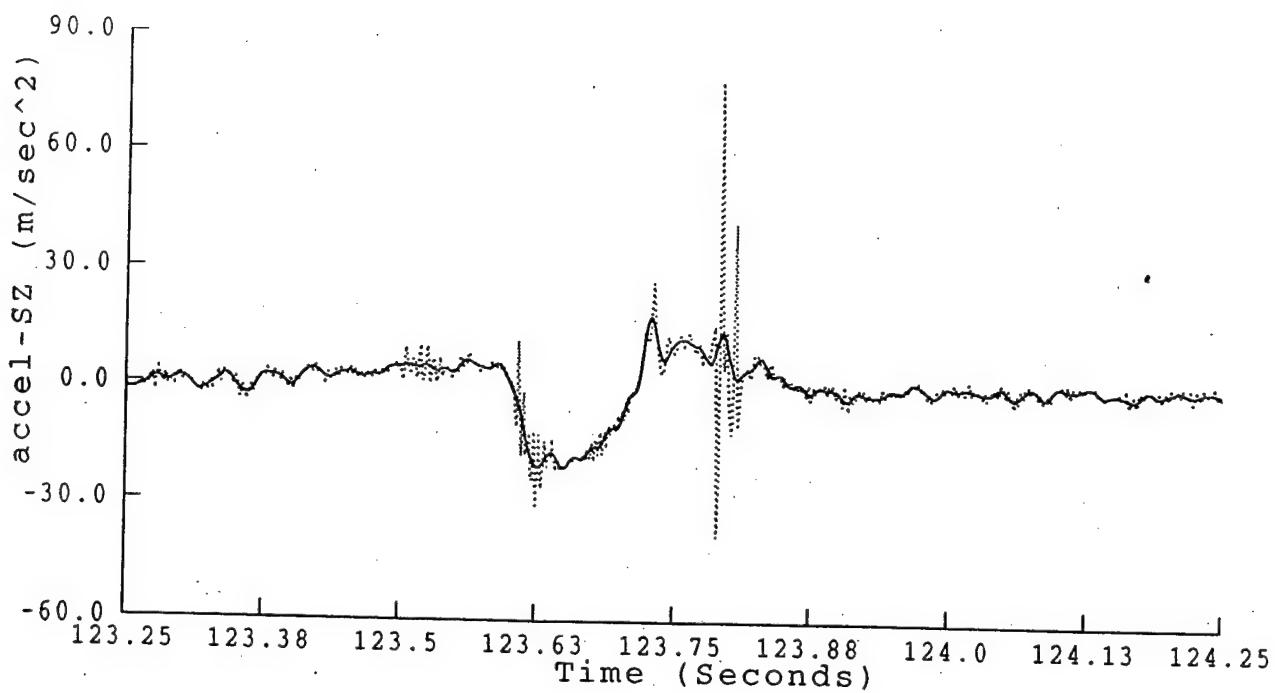
Figure 13.   Acceleration at the seat (Sz) for a -3 g, 4 Hz z axis shock. *Dotted line: unfiltered data; full line: band pass filtered at 0.5 Hz to 60 Hz.*
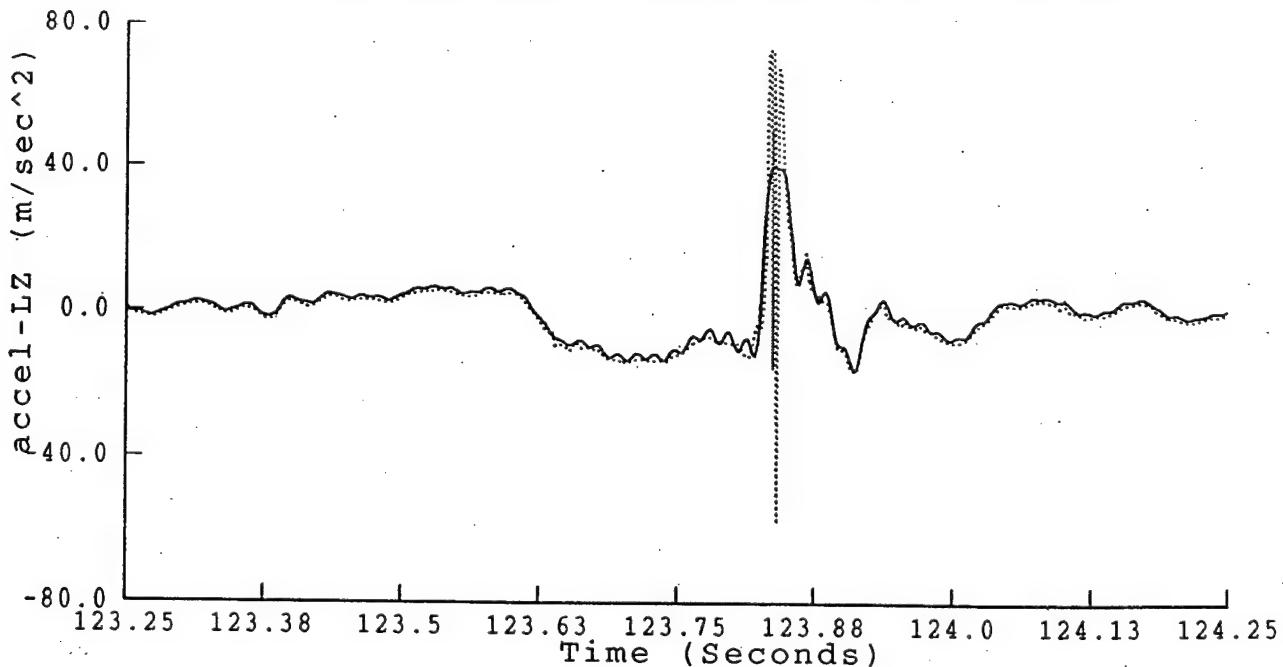
Figure 14.   Acceleration at the spine (L4 z) for a -3 g, 4 Hz z axis shock. *Dotted line: unfiltered data; full line: band pass filtered at 0.5 Hz to 60 Hz.*

133
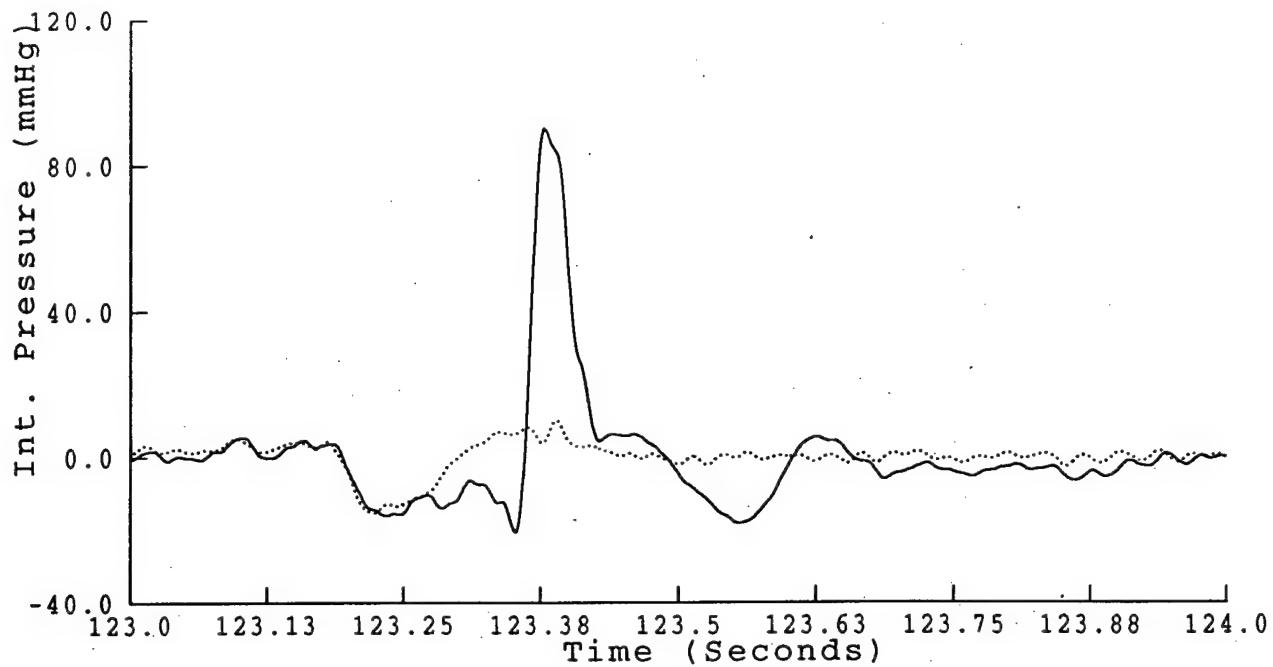
Figure 15.   Internal pressure measured for a -2 g, 4 Hz z axis shock. *Dotted line: seat Sz acceleration; full line: internal pressure.*
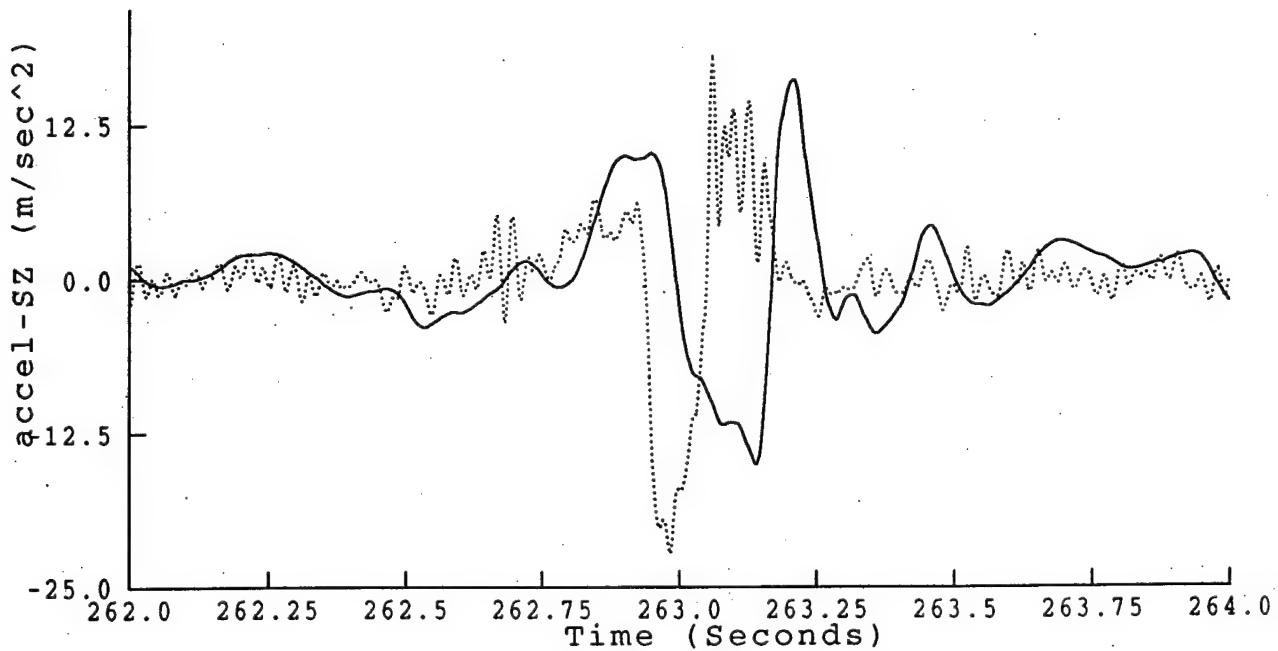
Figure 16.   Abdominal respitrace displacement for a -3 g, 4 Hz z axis shock. *Dotted line: seat Sz acceleration; full line: abdominal displacement.*

134
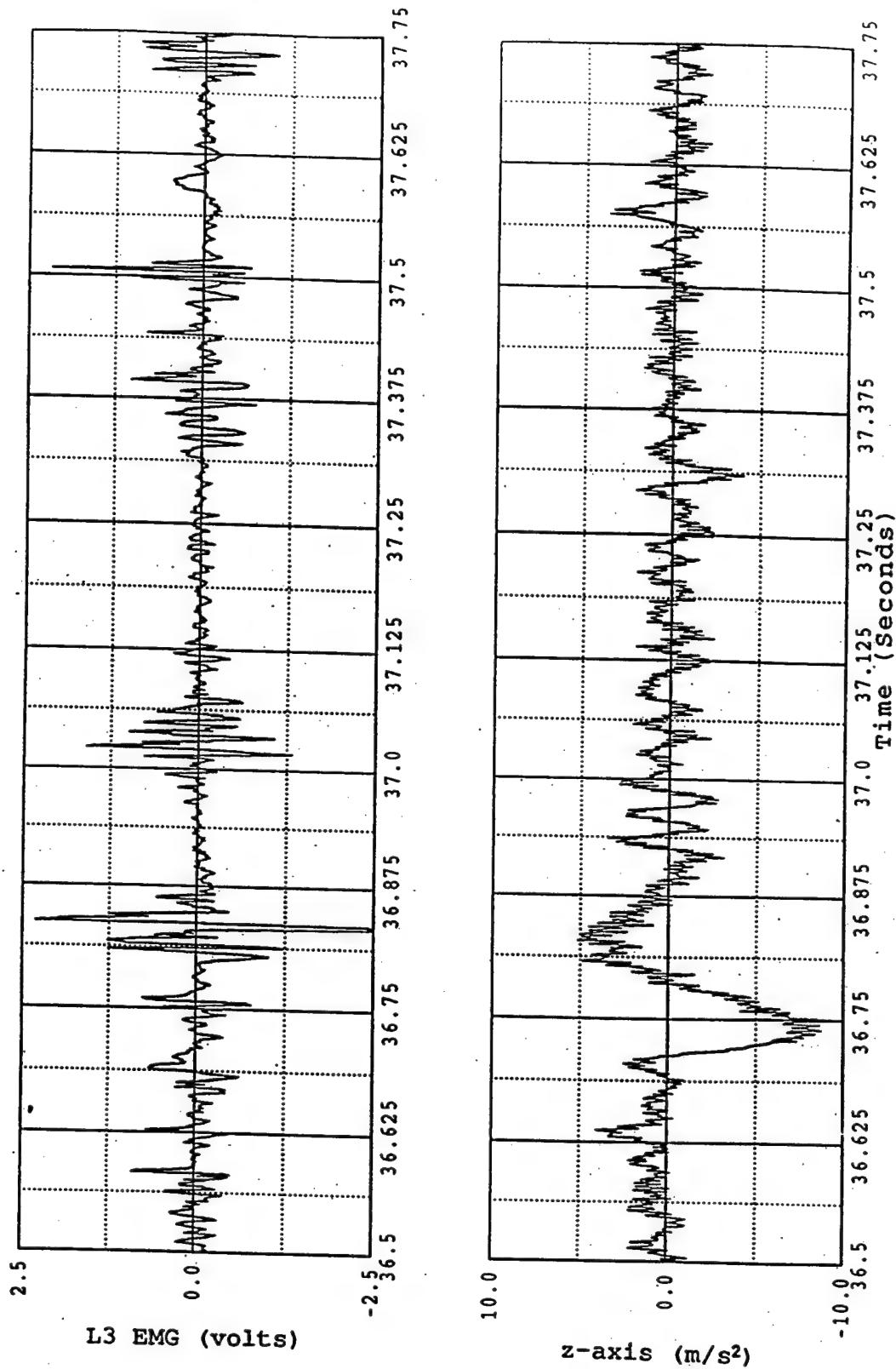
Figure 17   A typical response of lumbar muscle (volts) to a
             negative 1 g z axis shock at a frequency of 6 Hz.
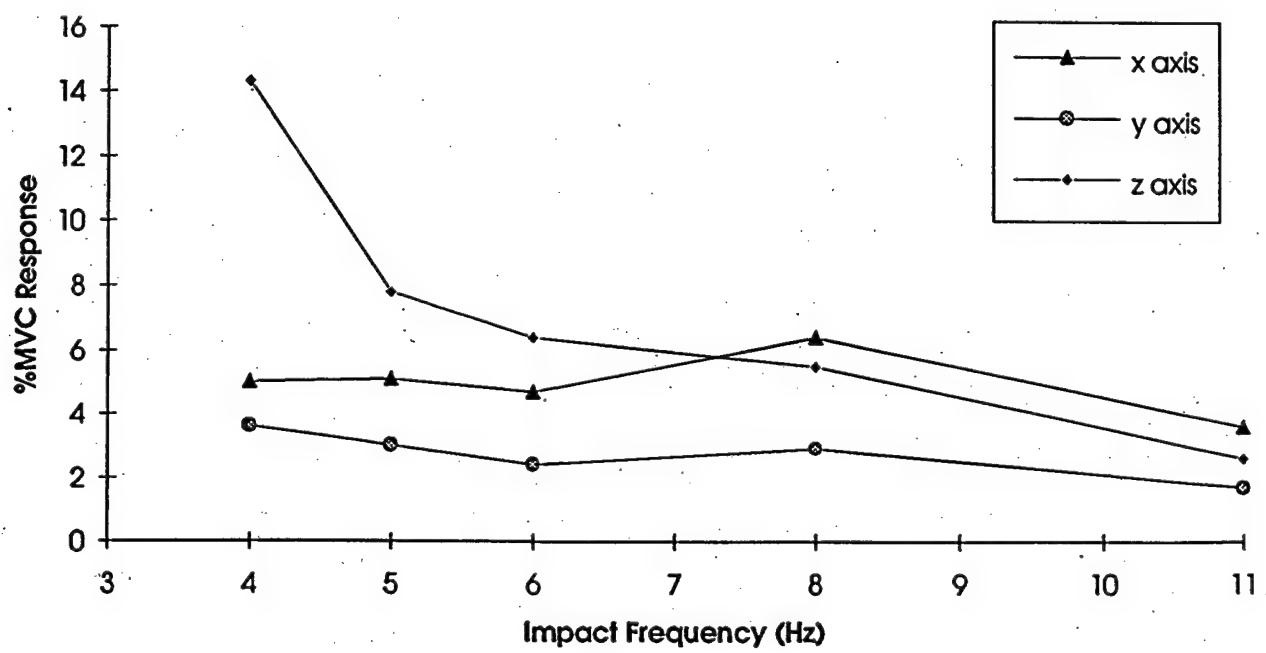
Figure 18. The mean response (n=20) of lumbar muscle to negative 3 g impact accelerations at frequencies of 4 to 11 Hz in x, y, and z axes.
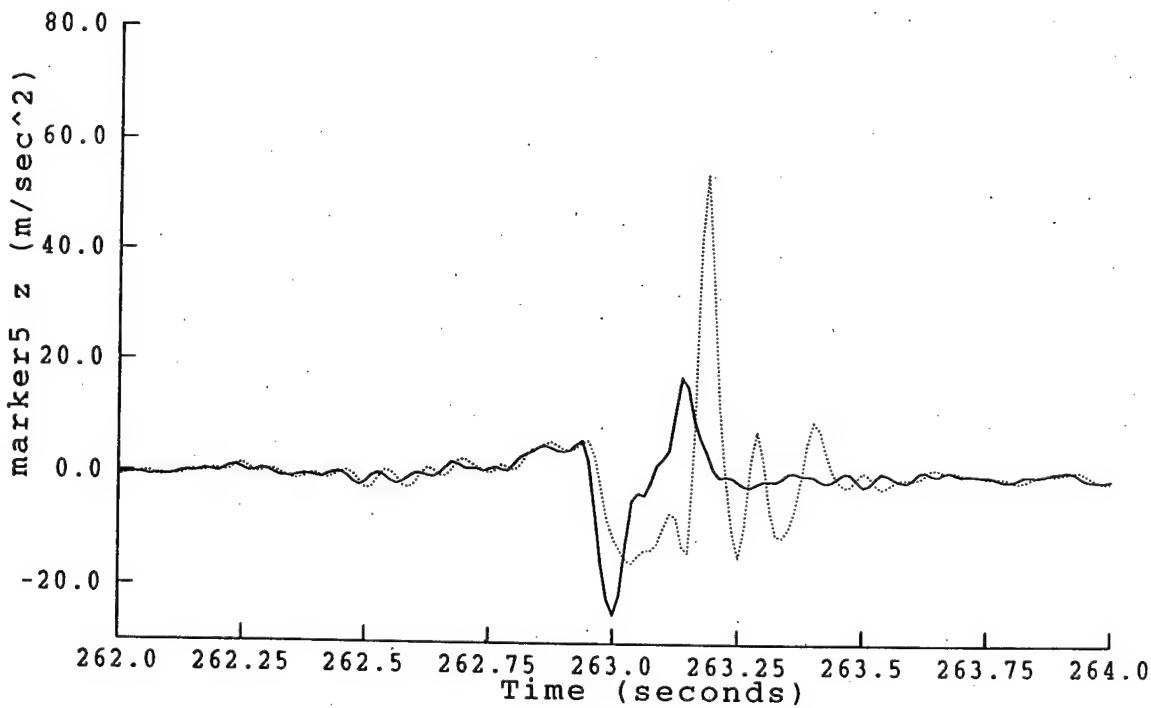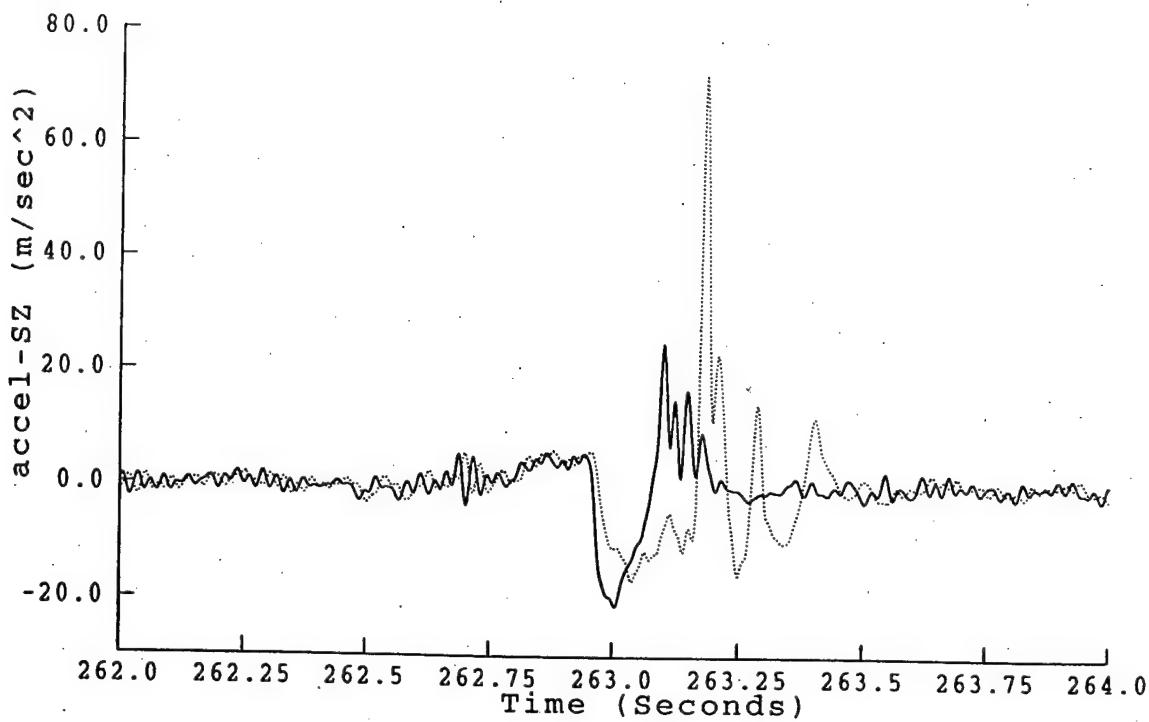
Figure 19.   Spinal response to a -3 g, 4 Hz z axis shock measured by accelerometer (top) and Optotrak (bottom). *Full lines: seat z; dotted lines: lumbar L4 z (top) and L5 z (bottom).*

137
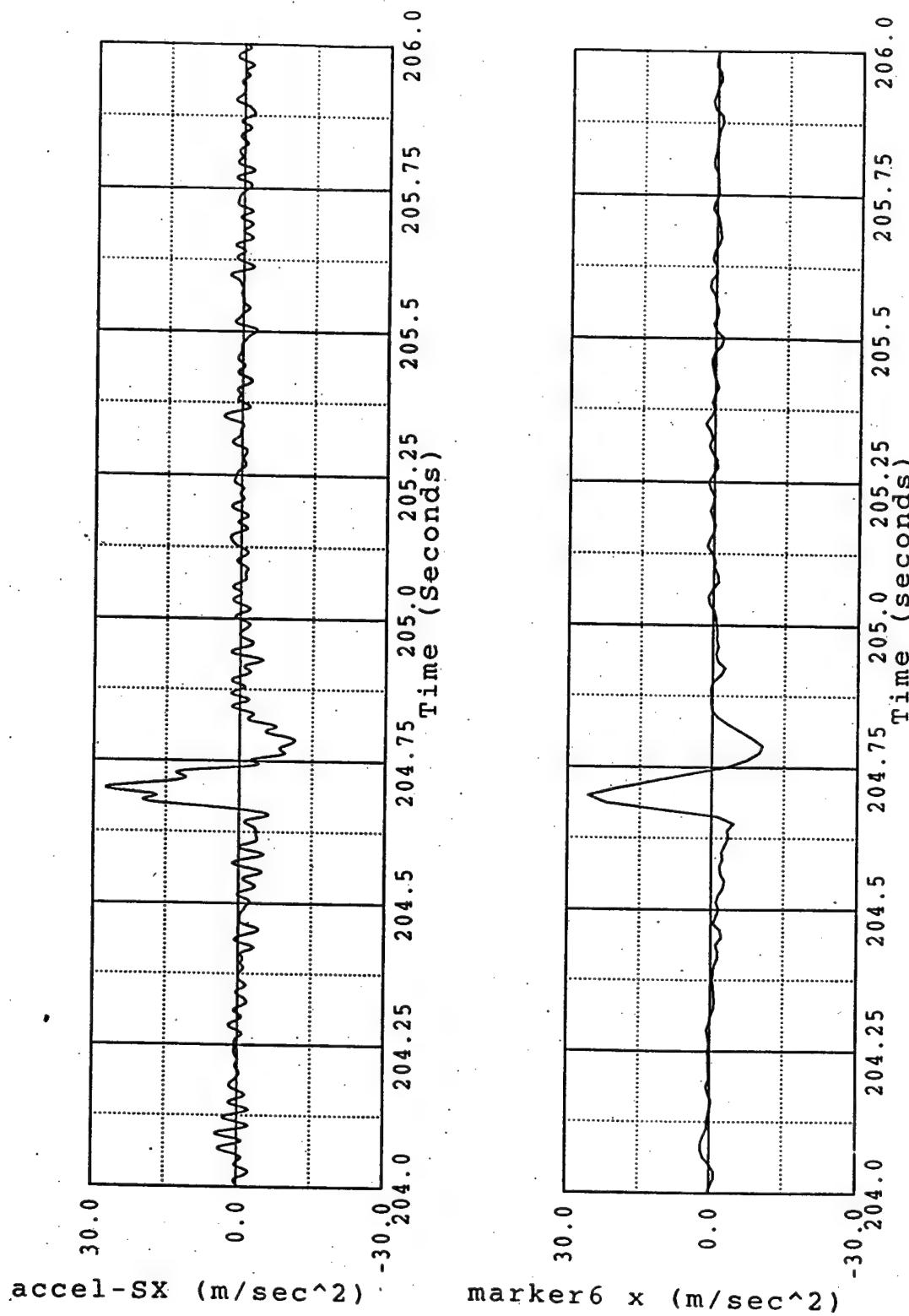
Figure 20.   3 g, 6 Hz x axis shock at the seat measured by accelerometer (top) and derived from Optotrak (bottom).
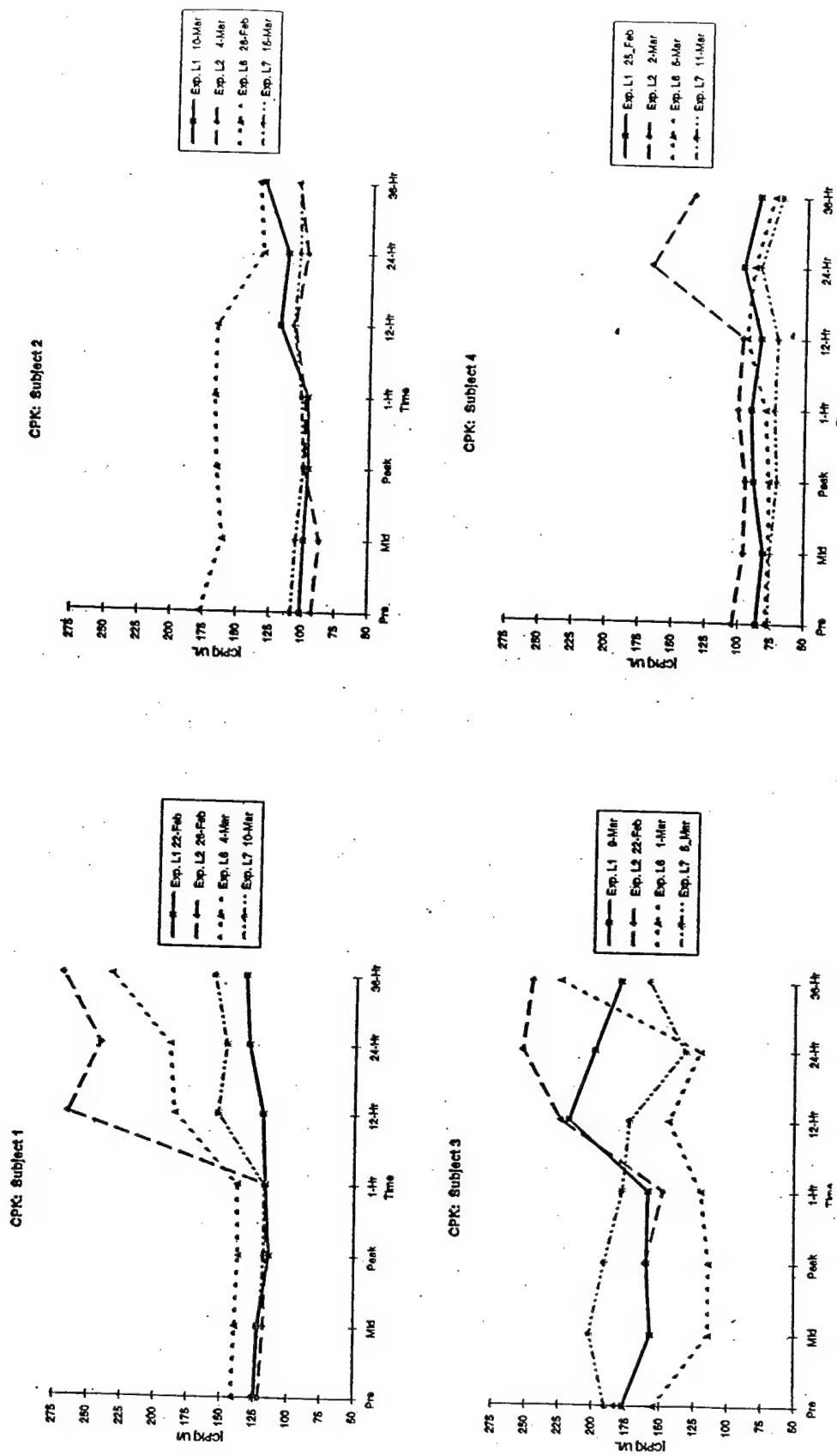
Figure 21. The individual response of creatine phosphokinase
(CPK) to a two hour exposure in four experimental
conditions in four subjects.
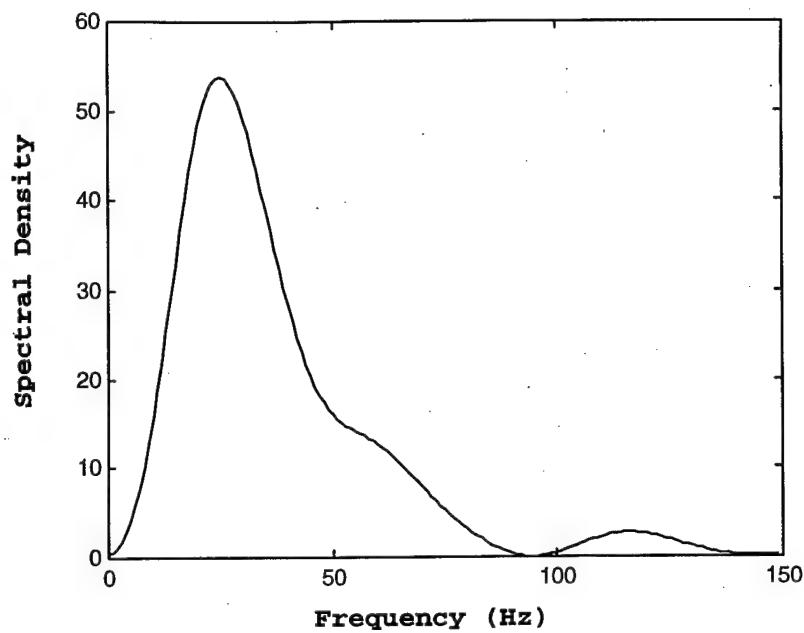
Figure 22.   Spectral density of a free damped oscillation of the skin-accelerometer system at L4 (z axis).
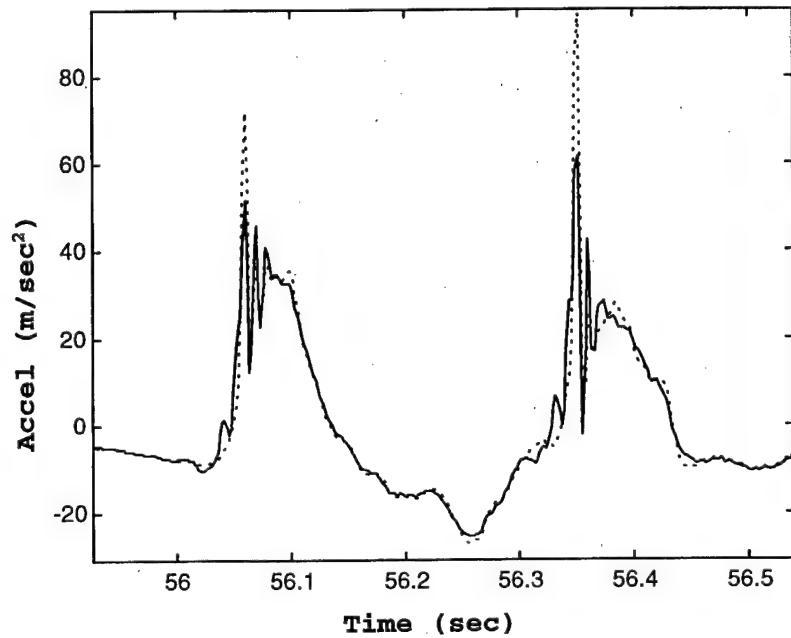
Figure 23.   Recorded L4 accelerometer response to a -4 g, z axis shock at the seat and the predicted acceleration at the spinous process after correction by the skin transfer function. Dotted line = recorded L4 response; Solid line = corrected response.

140
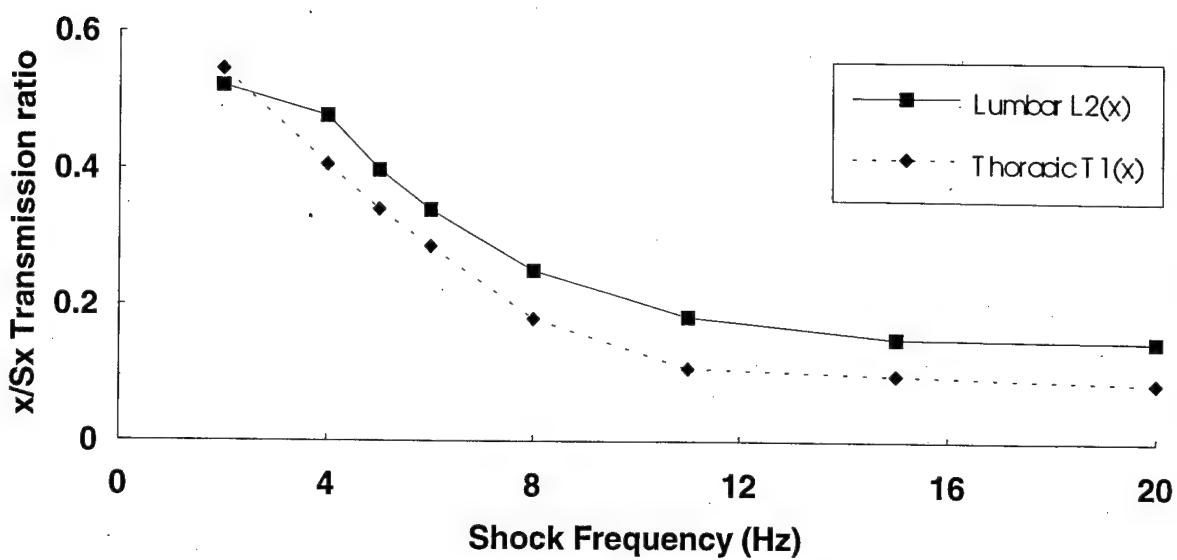
Figure 24.   Comparison of the mean transmission ratios of all
              shock amplitudes measured at the lumbar (L2) and
              thoracic (T1) spine in response to positive x
   axis shocks.
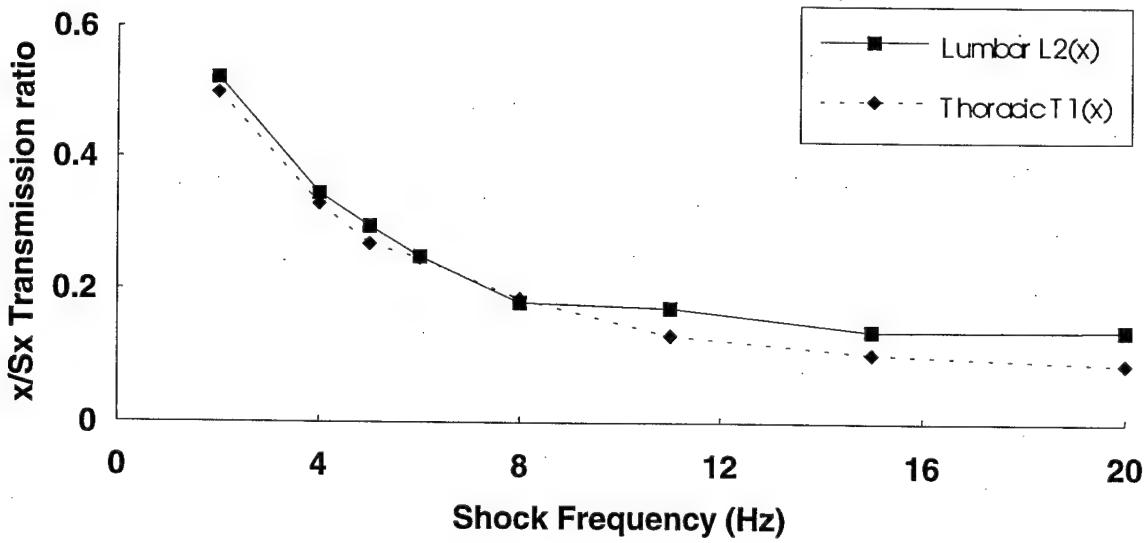
Figure 25.   Comparison of the mean transmission ratios of all
              shock amplitudes measured at the lumbar (L2) and
              thoracic (T1) spine in response to negative x
              axis shocks.

141
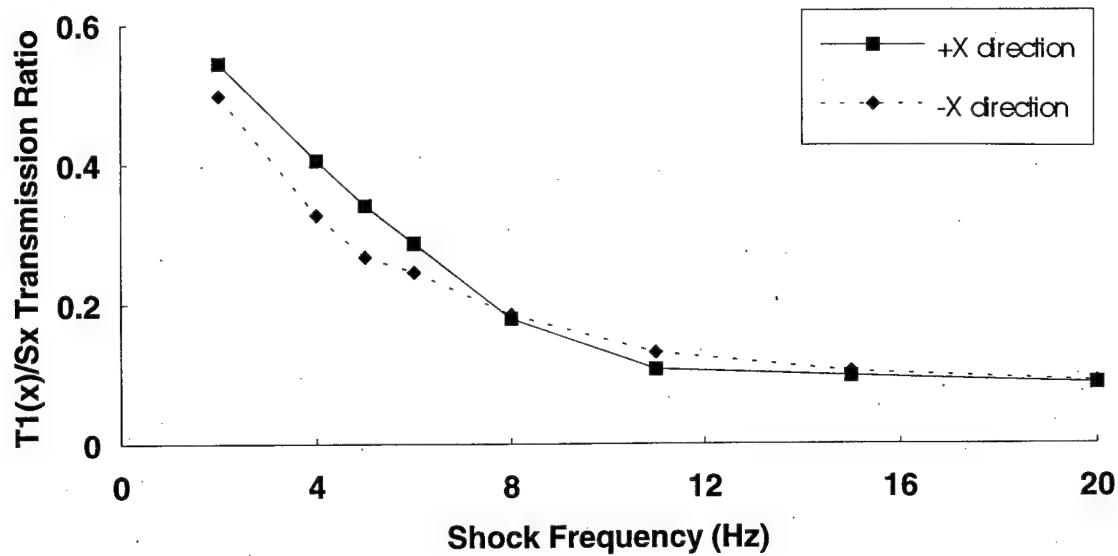
Figure 26. Comparison of the mean transmission ratios of all shock amplitudes measured at the lumbar (T1) spine in response to positive and negative x axis shocks.
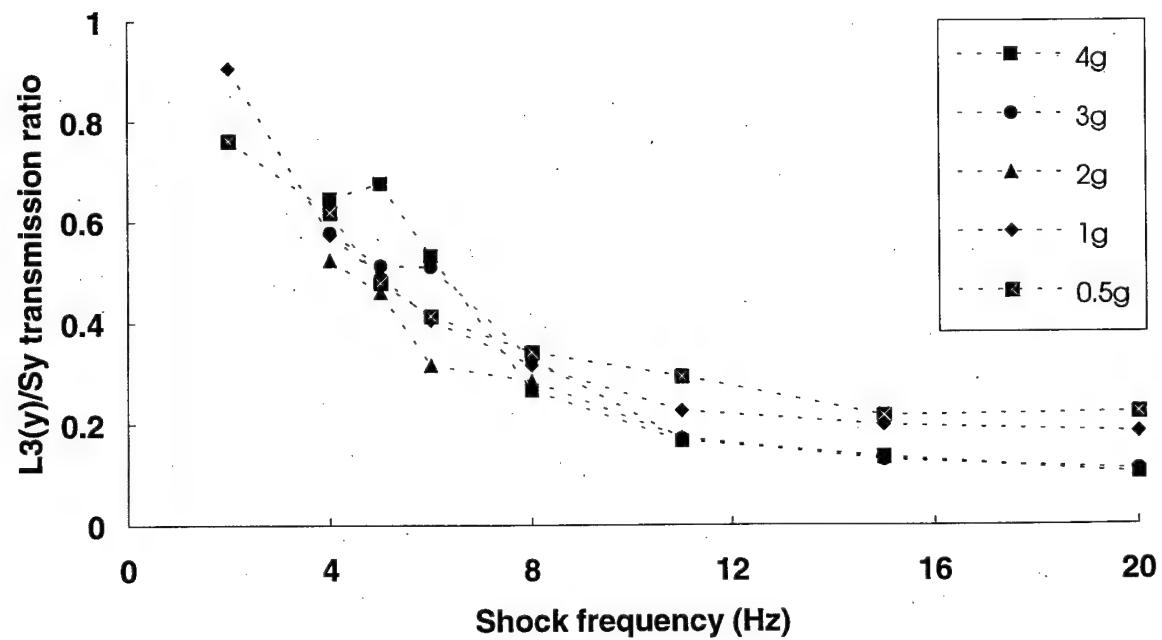
Figure 27. Spine (L3) y acceleration to seat y acceleration for 0.5, 1, 2, 3, 4 g shocks.

142
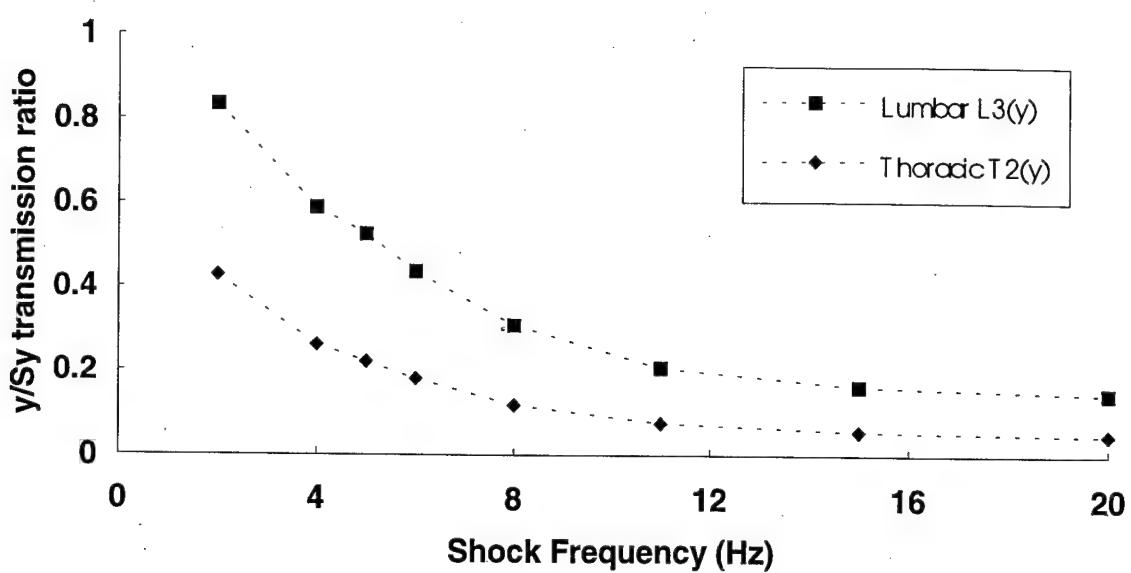
Figure 28.   Comparison of the mean transmission ratios of all
              shock amplitudes measured at the lumbar (L3) and
   thoracic (T2) spine in response to positive y
              axis shocks.
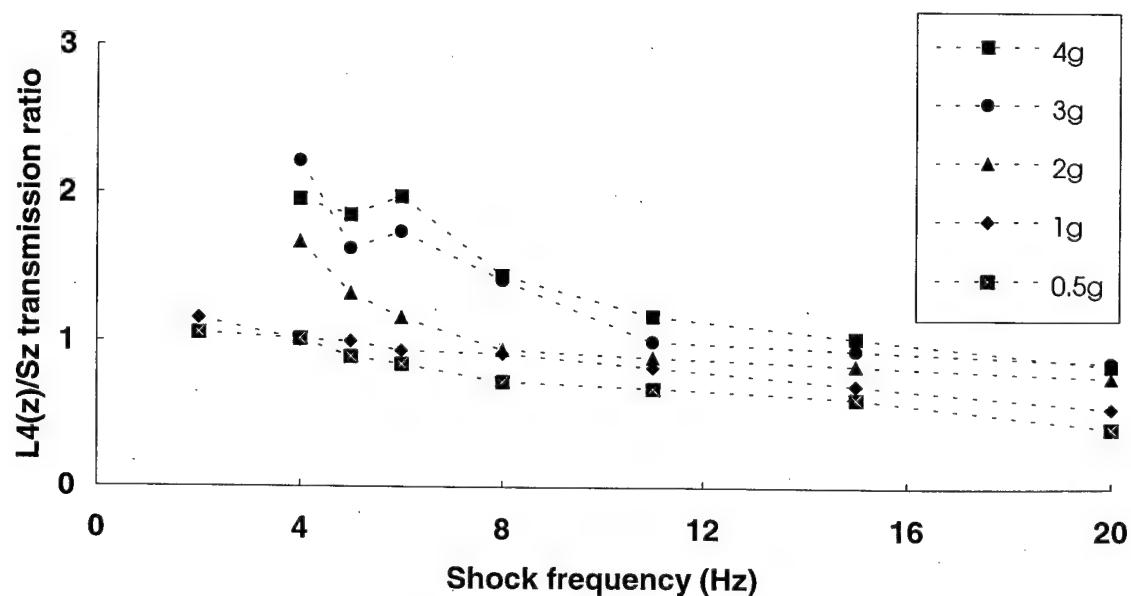
Figure 29.   Spine (L4) z acceleration to seat z acceleration
              for 0.5, 1, 2, 3, 4 g shocks.

143
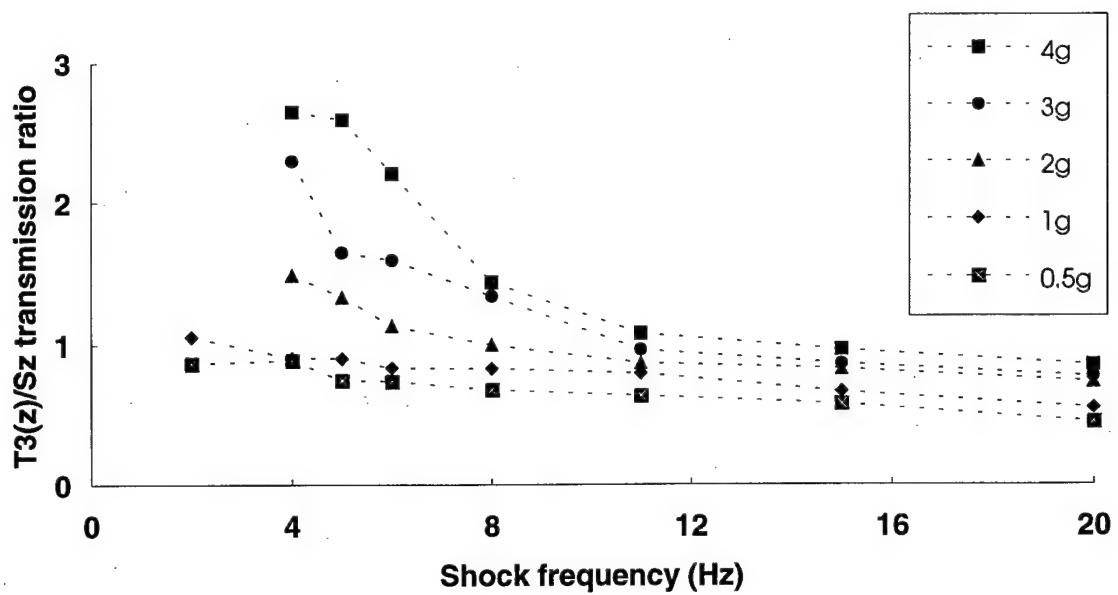
Figure 30.   Spine (T3) z acceleration to seat z acceleration
for 0.5, 1, 2, 3, 4 g shocks.
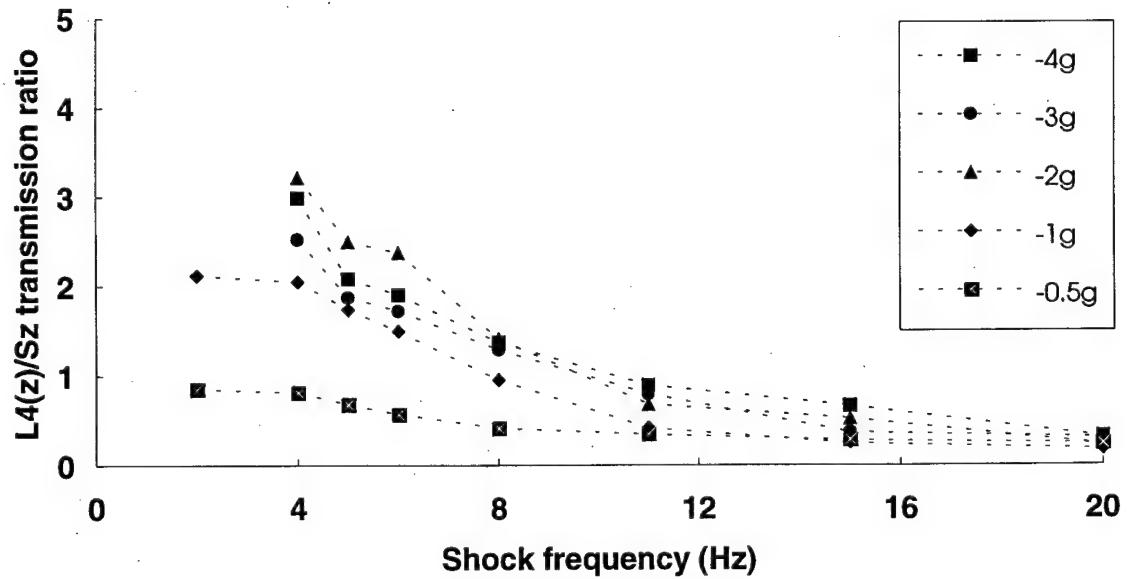
Figure 31.   Spine   (L4)   positive   z   acceleration   to   seat
z acceleration for -0.5, -1, -2, -3, -4 g shocks.

144
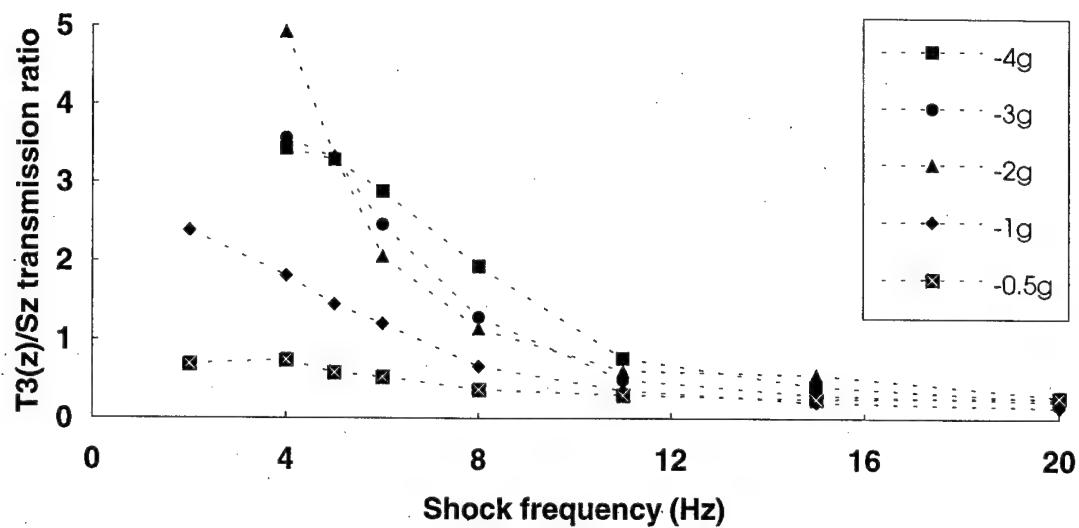
Figure 32.   Spine (T3) positive z acceleration to seat
z acceleration for -0.5, -1, -2, -3, -4 g shocks.
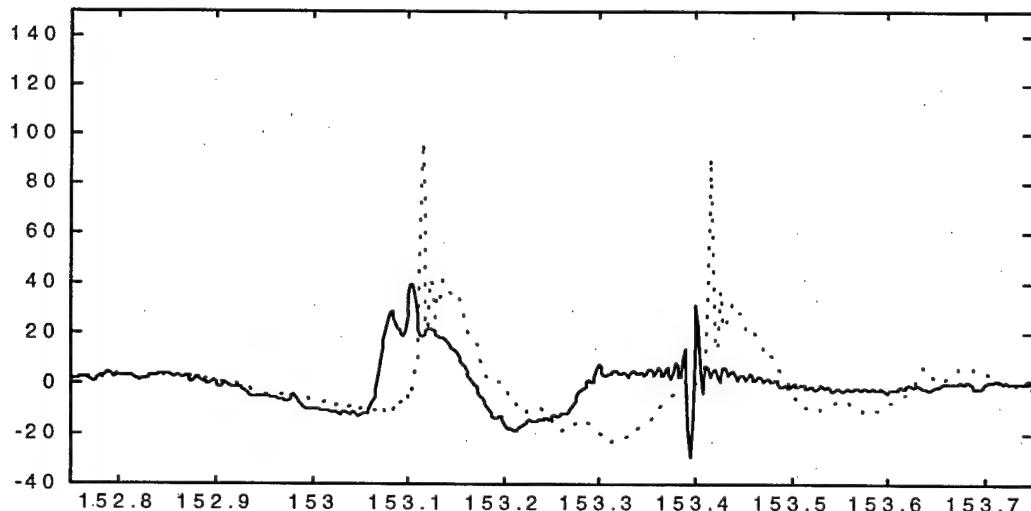

Figure 33.   Acceleration measured at the seat and lumbar
spine for a +4 g, 4 Hz z axis shock.   Dotted
line: Lumbar L4 z; solid line: Seat Sz.

145
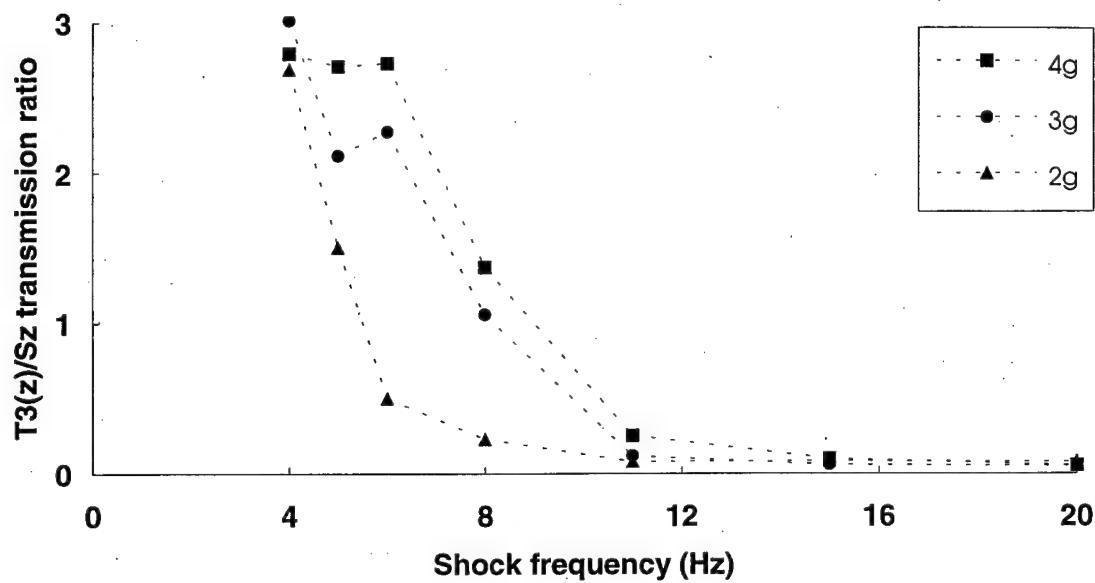
Figure 34.   Second peak of the spinal (T3) z acceleration response to seat z acceleration for positive 2, 3 and 4 g shocks.
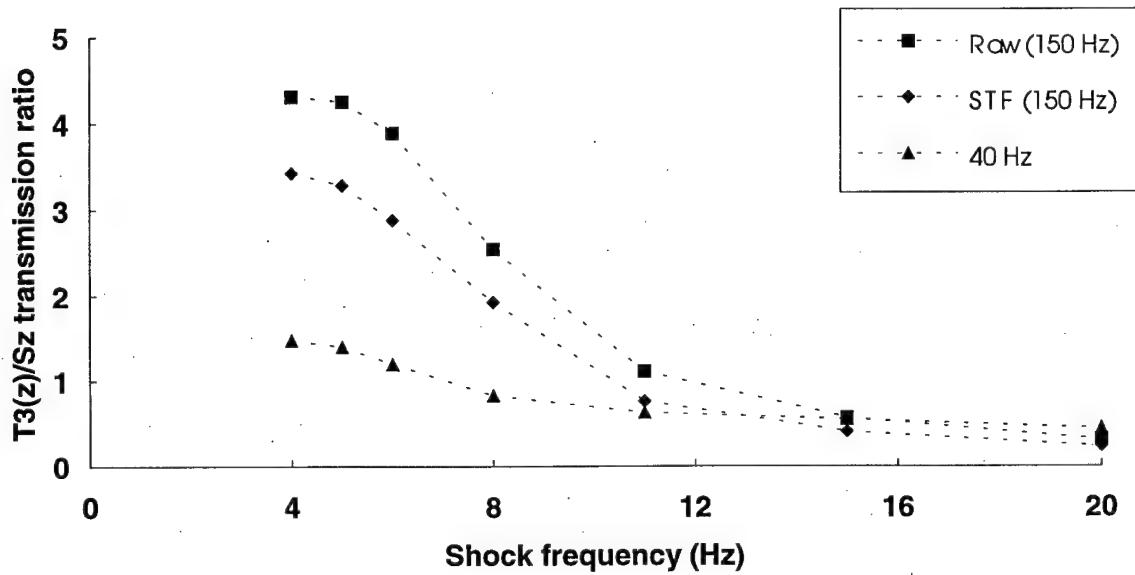

Figure 35.   Spine (T3) z acceleration to seat z acceleration for -4 g shocks using 40 Hz, 150 Hz (Raw), and Skin Transfer Function (STF) analysis.

146
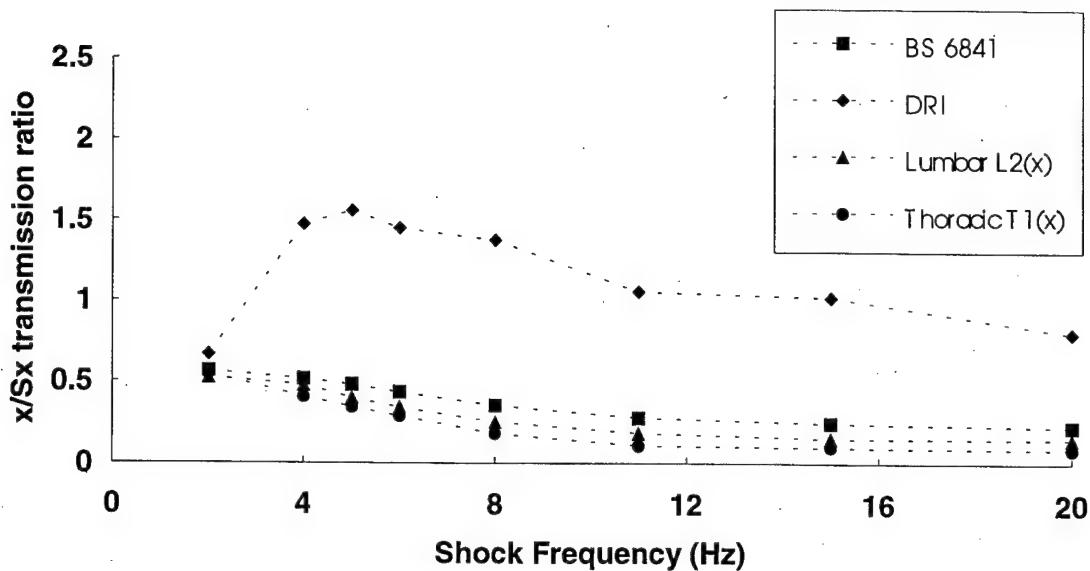
Figure 36.   Comparison of measured spine x transmission ratio
(positive shocks) with predicted transmission
ratios using BS 6841 filter and DRI models.
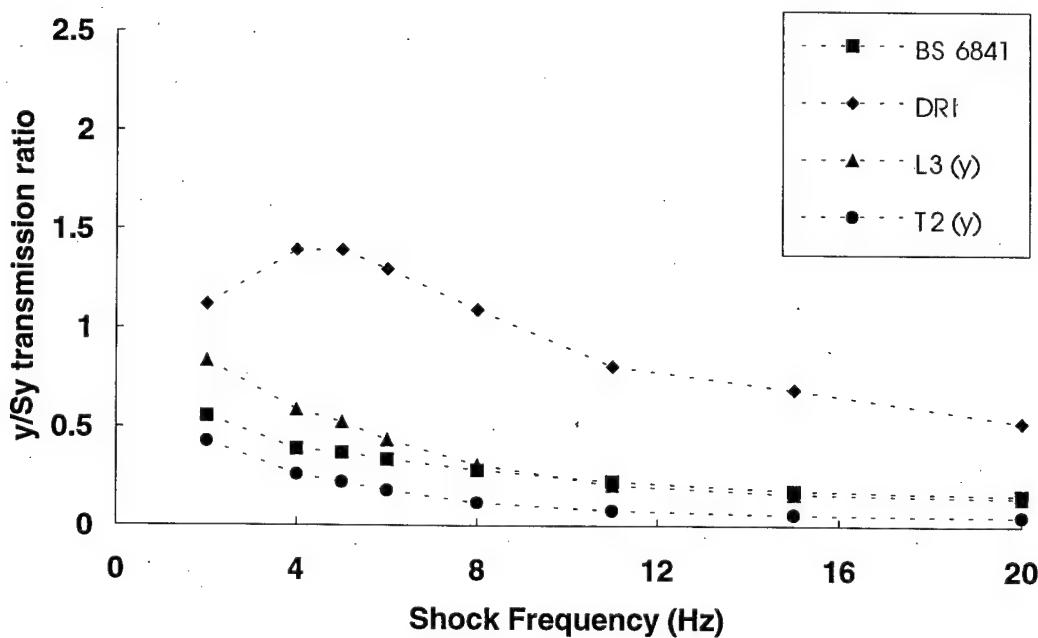
Figure 37.   Comparison of measured spine y transmission ratio
(positive shocks) with predicted transmission
ratios using BS 6841 filter and DRI model.
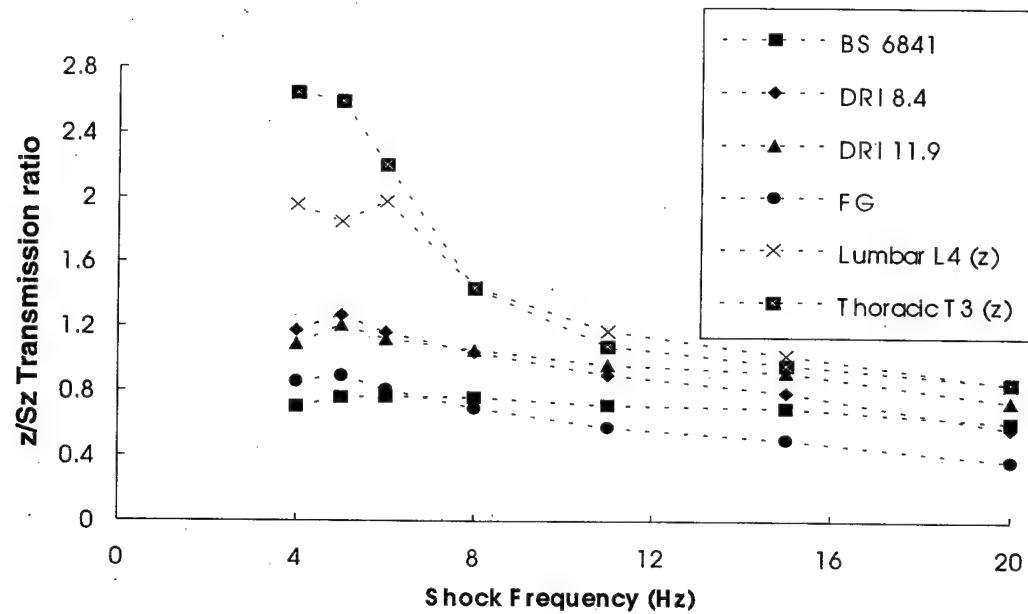
Figure 38.   Comparison of measured spine z transmission ratio
(4 g shocks) with predicted transmission ratios
using BS 6841 filter, Fairley-Griffin (FG) model
and DRI model.
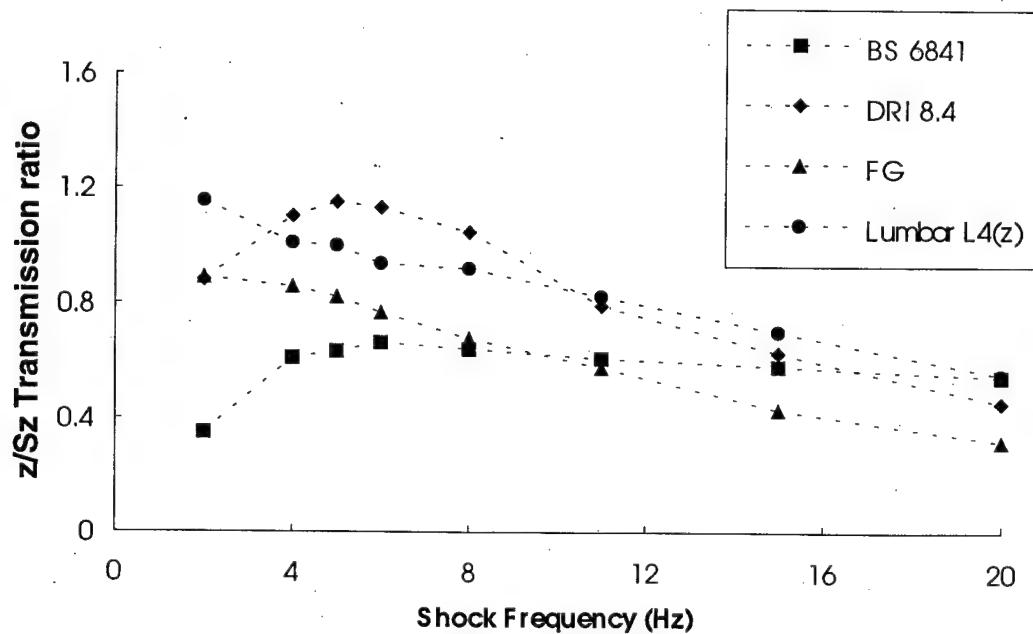

Figure 39.   Comparison of measured spine z transmission ratio
(1 g shocks) with predicted transmission ratios
using BS 6841 filter, Fairley-Griffin (FG) model
and DRI model.
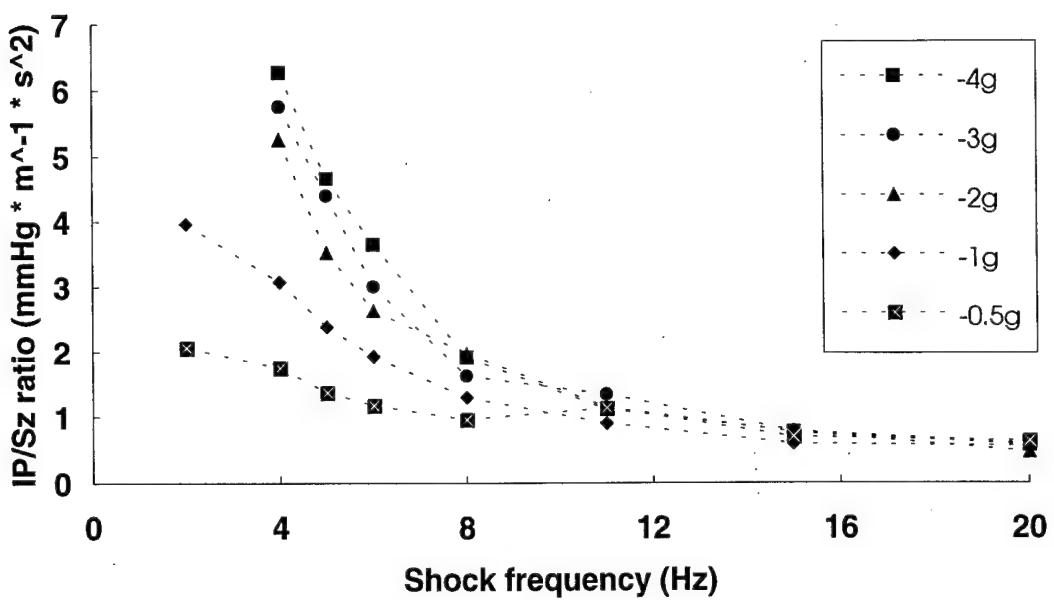
Figure 40.   Internal pressure response to seat z acceleration for -0.5, -1, -2, -3, and -4 g shocks.
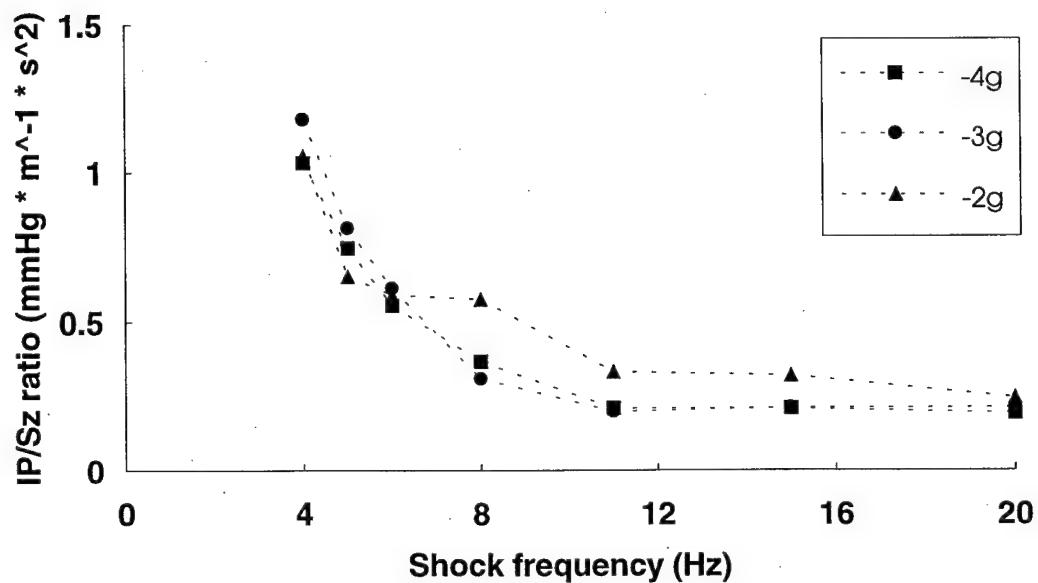
Figure 41.   Second internal pressure response to seat z acceleration for negative 2, 3 and 4 g shocks.

149
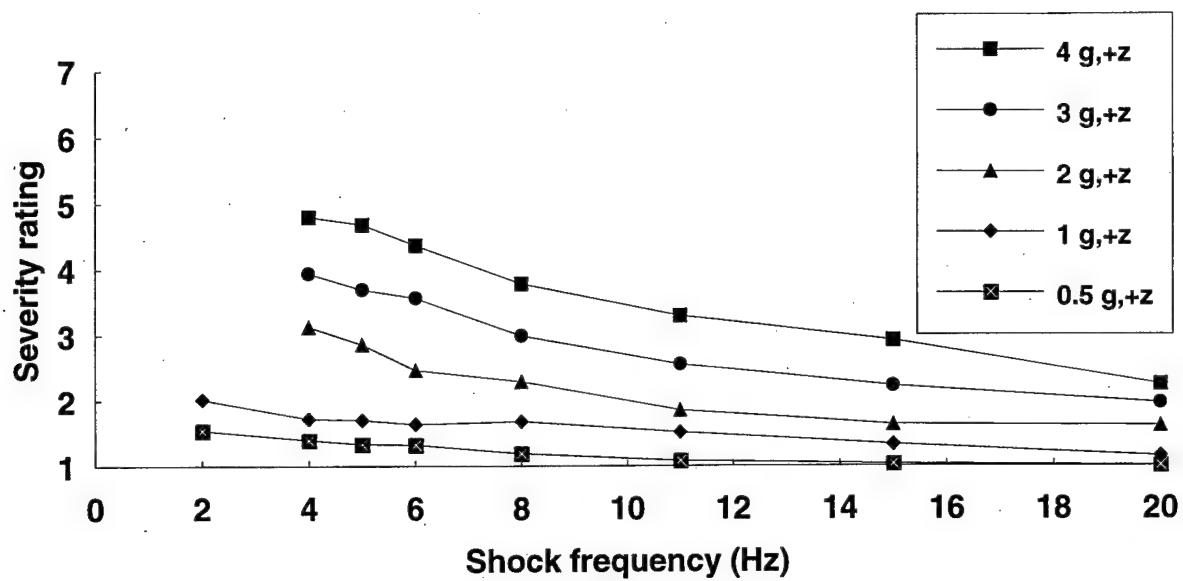
Figure 42.   Subjective severity ratings to single shocks in the positive z axis as a function of shock frequency and amplitude.
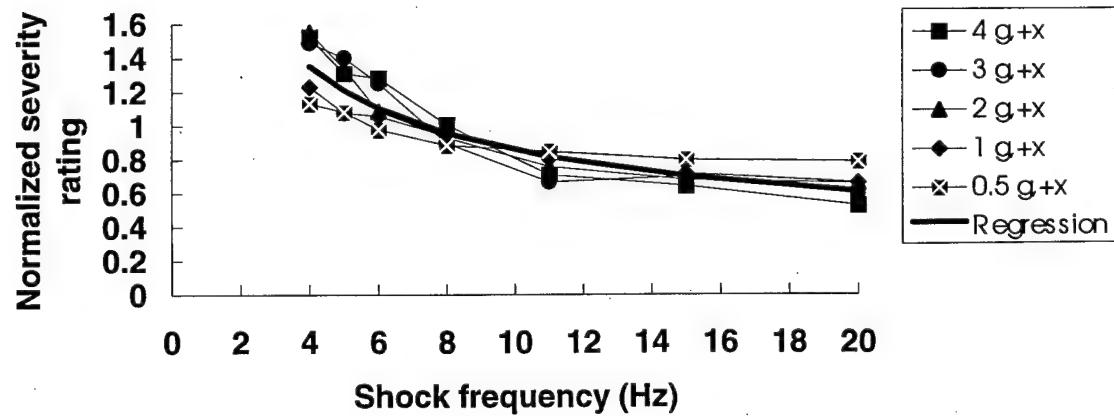
Figure 43.   Comparison of normalized subjective severity ratings to single shocks in the positive x axis for different shock magnitudes.   The solid line represents the regression line for all data.
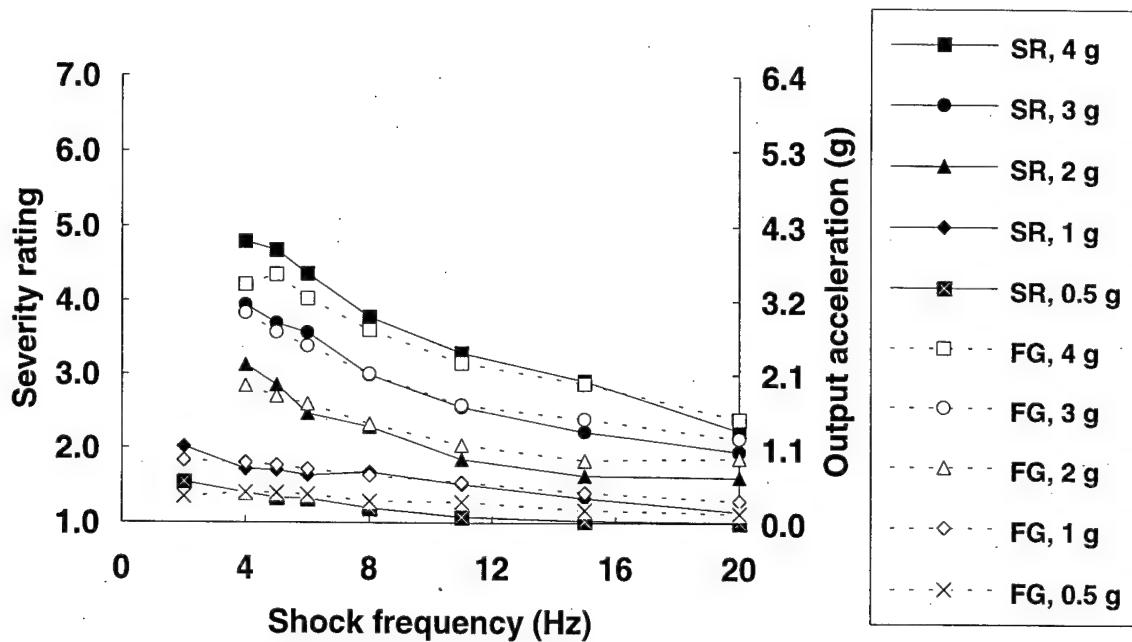
Figure 44.   Comparison between severity ratings (SR) and
expected output from the Fairley-Griffin (FG)
model to positive z axis shocks.
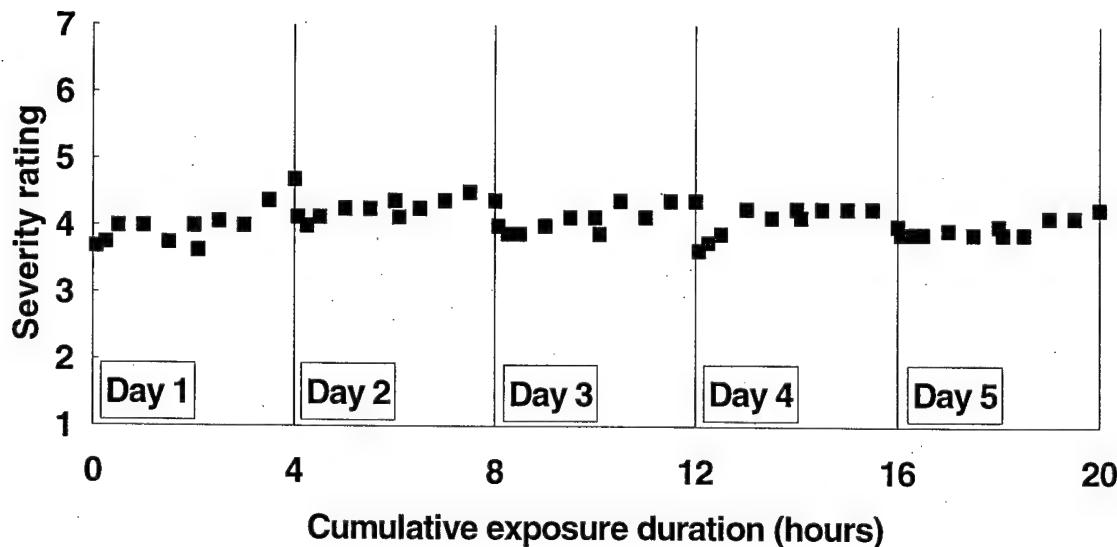
Figure 45.   Subjective severity ratings as a function of
cumulative exposure duration for 4 hour repeated
shock exposures in five consecutive days.

151
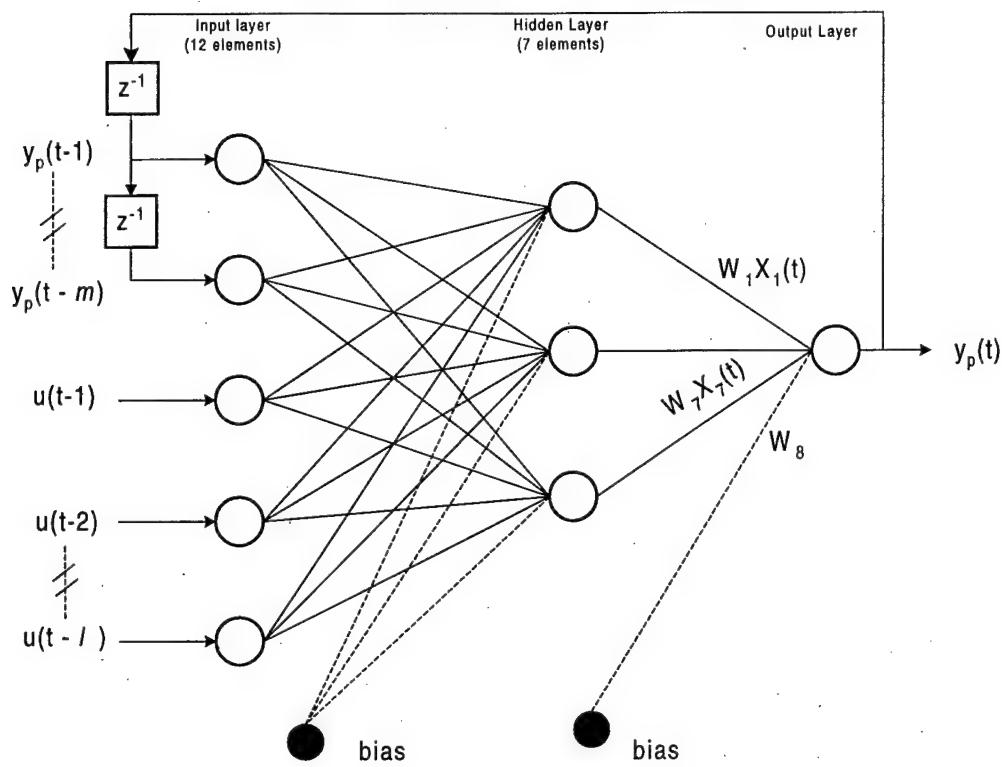
Figure 46.   Finalized neural network structure.
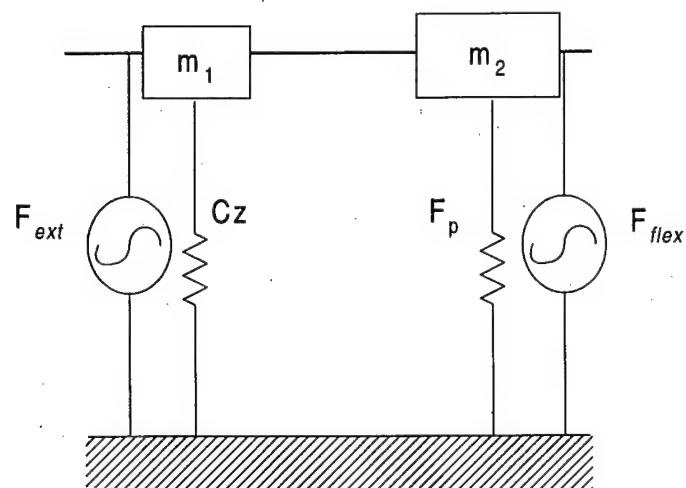
Figure 47.   The eccentric segregated mass model (ESMM).

152
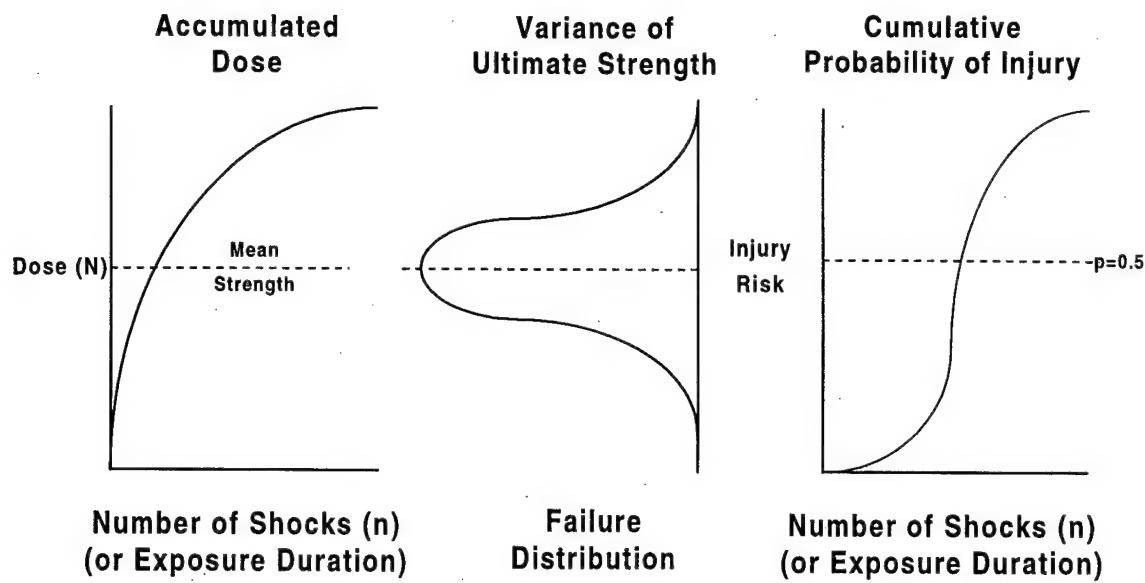
Figure 48. Cumulative probability function: relationship between acceleration dose and risk of injury.
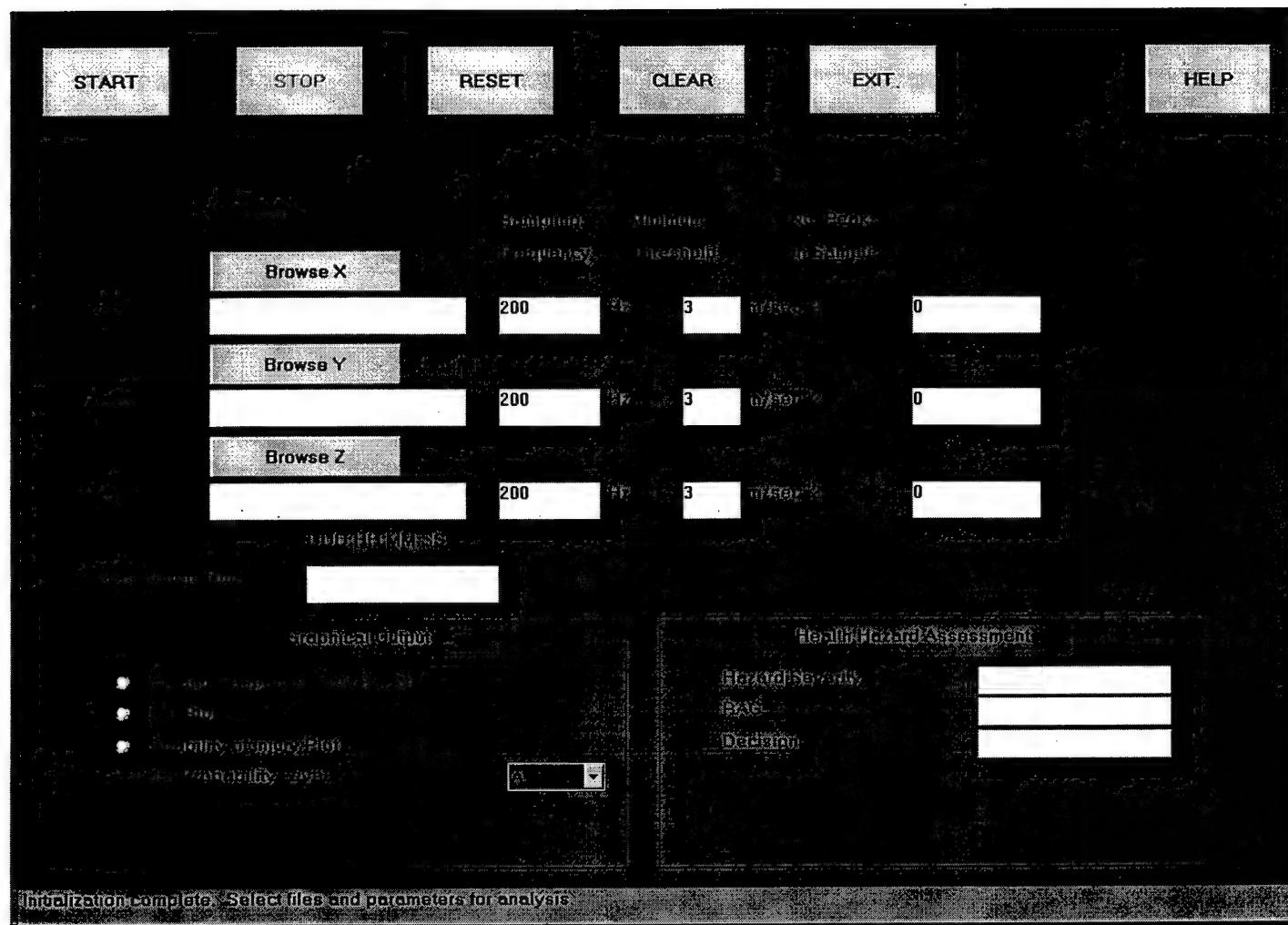
Figure 49.   Health hazard assessment (HHA) method graphical
user interface (GUI).
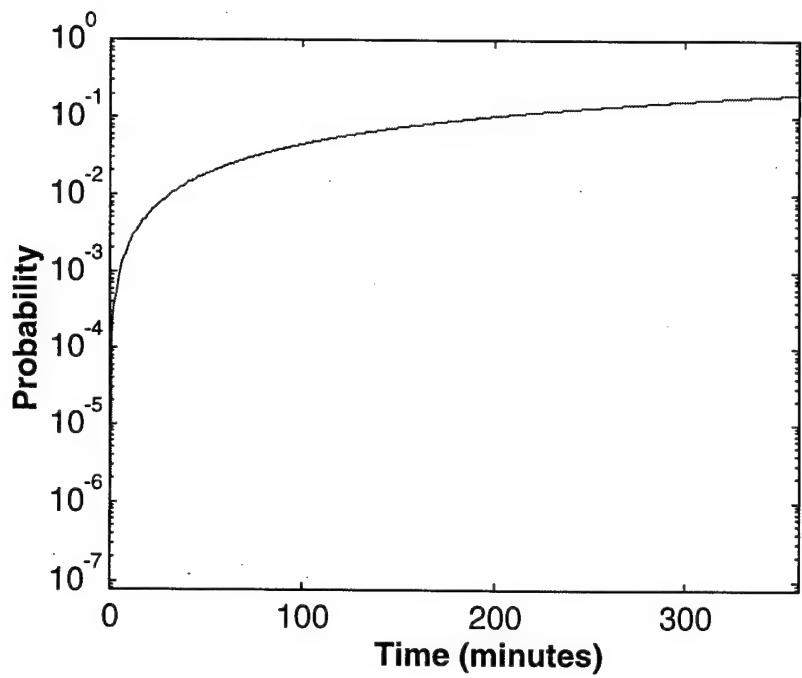
Figure 50. The risk of injury predicted for 6 hours of exposure to ±2 g and +4 g z axis shocks at the seat. Shock rate:32 ±2 g and 2 +4 g shocks per 5 min.

Appendix D

Equipment

The major equipment used during the experiments is listed below:

- Channel amplifiers and signal conditioning unit (Terrascience, Canada)

- Electrocardiograph: 12 lead (Marquette Electronics Inc., Model MAC15)

- Electrocardiograph: 3 lead (Hewlett Packard, Model 78304)

- Electromyograph: Telemg (Bioengineering Technology Systems, Milan, Italy)

- Entran miniature pressure transducer (Model EPB-140-5s)

- Force transducer (Maywood Instruments Ltd., Basingstoke, U.K., Model U4000 Load Cell)

- MARS Multiaxis ride simulator (Schenck/Pegasus 5900)

- Miniature accelerometers (9) range of $\pm10g$ and $\pm25g$ (EGAX-25, Entran Devices, N.J.)

- Optotrak Motion Analysis System (Northern Digital, Canada)

- PC computers

- Piezo-electric accelerometers (P.C.B. 301A03)

- Power supply (PCB 482A05)

- Respitrace monitor (Ambulatory Monitoring Inc., N.Y.)

- Seatpad to house triaxial accelerometer cluster

- Tape recorder (14 Channel analog recorder, TEAC XR510).

- VAX 4000/200 computer system

- Voltmeter

- Other general laboratory equipment and supplies

Appendix E

Publications based on Contract No. DAMD17-91-C-1115

To date, this work has resulted in a number of scholarly works which have been presented and published as papers in refereed journals, conference proceedings, post-graduate dissertations, and technical reports.

Journals

Nicol,J., Morrison, J.B., Roddan, G., and Rawicz, A. 1997. Modeling the Dynamic Response of the Human Spine to Shock and Vibration using a Recurrent Neural Network. International Journal of Vehicle Dynamics (in press).

Conference proceedings

Brammer, A.J., Roddan, G., Village, J., and Morrison, J.B. 1993. Machine identification of waveform characteristics, with application to seat motion. In Canadian Acoustical Association, October 8, Toronto, Canada.

Cameron, B.J., Robinson, D.G., Morrison, J.M., and Albano, J.P. 1995. Biochemical and EMG responses to extended exposure to mechanical shocks. In Human Response to Vibration, September 18-20, Bedford, England.

Morrison, J.B., Village, J., Roddan, G., Remedios, B., Robinson, D., Rylands, J., and Cameron, B. 1993. Analysis of spinal accelerations in response to 1, 2 and 3 g impacts at the seat. In Human Response to Vibration, September 20-22, Farnborough, England.

Morrison, J.B., Robinson, D., Roddan, G., Village, J., and Butler, B.P. 1994. Comparison of human response to impacts measured by infrared emitting diodes and accelerometers. In International Ergonomics Association (IEA), August 15-19, Toronto, Canada.

Morrison, J.B., Robinson, D.G., Roddan, G., Nicol, J.J., and Butler, B.P. 1995. Analysis of vertebra to skin transfer function in response to mechanical shock. In Human Response to Vibration, September 18-20, Bedford, England.

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Morrison, J.B., Martin, S.H., Robinson, D.G., Roddan, G., Nicol, J.J., Springer, M.J-N., Cameron, B.J., and Albano, J.P. 1996. Development of a comprehensive method of Health Hazard Assessment (HHA) for exposure to repeated mechanical shocks. In Human Response to Vibration, September 18-20, Nuneaton, England.

Nicol, J.J., Morrison, J., Roddan, G., and Rawicz, A. 1995. The application of an artificial neural network to modeling seat to spine transmission of acceleration. In Human Response to Vibration, September 18-20, Bedford, England.

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Robinson, D., Morrison, J.B., and Village, J. 1993. The pattern of electromyographic response to mechanical shocks. In Human Response to Vibration, September 20-22, Farnborough, England.

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Robinson, D., Brown, D., Morrison, J.B., Cameron, B., and Village, J. 1994. A method to quantify the paraspinal muscle response to impacts at the seat. In International Ergonomics Association (IEA), August 15-19, Toronto, Canada.

Robinson, D.G., Morrison, J.M., and Cameron, B.J. 1995. The contribution of muscle response and internal pressure to estimation of spinal compression from mechanical shocks using a simple biomechanical model. In Human Response to Vibration, September 18-20, Bedford, England.

Rylands, J., Remedios, B., Morrison, J.B., and Village, J. 1993.
A method for processing ECG signals to assess the
instantaneous effects of mechanical shocks. In Human Response
to Vibration, September 20-22, Farnborough, England.

Village, J., and Morrison, J.B. 1991. Development of a standard
for the Health Hazard Assessment of mechanical shock and
repeated impact. In Human Response to Vibration, September
25-27, Buxton, England.

Village, J., Rylands, J., and Morrison, J.B. 1993. Development of
a dose-effect database for exposure to whole-body vibration:
problems and assumptions. In Human Factors Association of
Canada, August, Halifax.

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Technical reports

Cameron, B., Morrison, J., Robinson, D., Vukusic, A., Martin, S.,
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Hazard Assessment of Mechanical Shock and Repeated Impact in
Army Vehicles, Phase 4 - Experimental Phase. Fort Rucker, AL:
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Village, J., Morrison, J., Robinson, D., Roddan, G, Rylands, J., Cameron, B., Remedios, B., and Brown, D. 1995b. <u>Development of a Standard for the Health Hazard Assessment of Mechanical Shock and Repeated Impact in Army Vehicles, Phase 3 Pilot Tests</u>. Fort Rucker, AL: USAARL Contract Report No. CR 95-3.

Theses

Nicol, J.J. 1996. Modeling the dynamic response of the human spine to mechanical shock and vibration using an artificial neural network. Master's thesis, Simon Fraser University.

Robinson, D.G. 1997. The Dynamic Human Response to Seated Shocks: The Influence of Muscle and Internal Pressure. Ph.D. dissertation, Simon Fraser University.

Vukusic, A.V. 1995. Subjective and associated objective response to repeated mechanical shocks in seated humans. Master's thesis, Simon Fraser University.

Appendix F

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A full list of references complied in the Phase 1 literature review is available in the Phase 1 Report, July 15, 1992.

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162

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163

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Appendix G

Glossary

Δ_{max}	maximum change
ϖ_n	natural frequency
δ	amplitude
$^\circ$	degrees
Φ	probability of injury
σ	variance
σ_μ	static failure stress
σ_q	applied stress level
ς	fraction of critical damping ratio
$\neq$	not equal to
$\pm$	plus or minus
$+x$	positive x axis vibration or shock according to biodynamic convention: forward (ISO 2631,1985)
$+y$	positive y axis vibration or shock according to biodynamic convention: to left (ISO 2631,1985)
$+z$	positive z axis vibration or shock according to biodynamic convention: upward (ISO 2631,1985)
$-x$	negative x axis vibration or shock according to biodynamic convention: backward (ISO 2631,1985)
$-y$	negative y axis vibration or shock according to biodynamic convention: to right (ISO 2631,1985)
$-z$	negative z axis vibration or shock according to biodynamic convention: downward (ISO 2631,1985)

%	percent
>	greater than
<	less than
ASCC	Air Standardization Coordinating Committee
a_w	frequency weighted acceleration
BS	British Standards
C	critical damping ratio
C1	first cervical vertebrae
C7	seventh cervical vertebrae
CPK	creatine phosphokinase
Cx	anterio-posterior shear forces at L4/15 joint
Cy	lateral shear forces at L4/15 joint
Cz	compressive forces at L4/15 joint
Cz_i	compressive force obtained from biomechanical model
Cz_u	ultimate compressive strength of lumbar L4/L5 joint
Cz_e	compressive force applied
D	generalized dose function
DRI	dynamic response index
DRI_0	estimated static failure stress
ECG	electrocardiography
EMG	electromyography
F	degree of fatigue
FAV	Fast Attack Vehicle

FFT	fast Fourier transform
fn	natural frequency
g	acceleration due to gravity (9.81 m.s^{-2})
GI	gastro-intestinal
GUI	graphical user interface
Hb	Hemoglobin
HHA	health hazard assessment
Hz	Hertz
IEMG	integrated electromyography
IRED	infra-red emitting diodes
ISO	International Organization for Standardization
i	shock
L1	first lumbar vertebrae
L2	second lumbar vertebrae
L3	third lumbar vertebrae
L4	fourth lumbar vertebrae
L5	fifth lumbar vertebrae
L/day	litres per day
LDH	lactate dehydrogenase
LL	left lumbar
LT	long term
LT1	Long-term experiment 1
LT2	Long-term experiment 2
LT3	Long-term experiment 3

LT4	Long-term experiment 4
LT5	Long-term experiment 5
MARS	Multiaxis ride simulator
MF	mean frequency
MIL-STD	Military standard
Mg^{2+}	magnesium ion
$m \cdot s^{-2}$	meters per second squared
MVC	Maximum voluntary contraction
Na^+	sodium ion
n_i	number of cycles completed at stress Si
N_i	number of cycles required to cause failure
n_q	observed number of impacts
N_q	maximum number of impacts
NIOSH	National Institute for Occupational Safety and Health
RAC	risk assesment code
RBC	red blood cells (erythrocytes)
rmd	tenth power root mean
rmq	root-mean-quad
rms	root-mean-square
RNN	recurrent neural network
R-R interval	interval between two consecutive heart beats on an electrocardiogram
RT	right thoracic
S.D.	standard deviation

S_e	static stress level
S_i	applied repetitive stress level
S_u	static failure stress
SOP	Standard Operating Procedure
SSM	simplified spine model
ST	short term
STMCB	Steiglitz-McBride parametric modeling method
ST1	Short-term experiment 1
Sx	seat acceleration in the x axis
Sy	seat acceleration in the y axis
Syn Work	Synthetic Work
Sz	seat acceleration in the z axis
T	extended period of time
t	time
T1	first thoracic vertebrae
T3	third thoracic vertebrae
T4	fourth thoracic vertebrae
T6	sixth thoracic vertebrae
T8	eighth thoracic vertebrae
T9	ninth thoracic vertebrae
T10	tenth thoracic vertebrae
TC	Technical Commitee
TGV	tactical ground vehicles
TTS	temporary threshold shift

USAARL	United States Army Aeromedical Research Laboratory
USAMRDC	United States Army Medical Research Command
VDV	vibration dose value
vWF	von Willebrand's factor
WBC	white blood cells
WBV	whole-body vibration
W.E.S.	Waterways Experimental Station